AUSCULTATION
OF THE
HEART

AUSCULTATION OF THE HEART

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To the Reader

You *must* be synesthetic
And really quite poetic
In reading *Auscultation of the Heart*

You must picture every murmur
Very weak or slightly firmer
And listen to the beauties of a chart

You must learn to be symbolic
Positively diabolic,
Hearing murmurs diastolic
Pre and mid and post systolic
Sometimes very melancholic
Sometimes full of fun and frolic

Screeching like a sea gull
Falling soft as snow
Regular arrhythmic
High in pitch or low

You need musical notation
Marking all the syncopation
Each and every palpitation
Listening with concentration
And with ocular observation
In this book called *Auscultation of the Heart*

MARJORIE KIMMERLE

Preface

FEW PHASES OF physical examination as important and as universally used as cardiac auscultation are done with so little confidence All doctors except for a small number of specialists have occasion to listen to the heart and yet few have learned to perform a skillful auscultation and to interpret properly the results To be sure some persons cannot adequately hear sounds and distinguish rhythms and consequently cannot listen satisfactorily to a heart But I believe that with an understanding of the basic principles of auscultation and with moderate experience most doctors can get the information made available by this procedure—and with the simplest of instruments

Auscultation often yields the first and only evidence of heart disease It is the most valuable means of recognizing valvular damage and enables the doctor to anticipate future changes in the heart and to treat the patient accordingly Most of the common arrhythmias can be recognized by auscultation alone although the electrocardiogram often must give the final answer Congenital heart deformities a failing myocardium pericarditis abnormal vascular pressures all give important auscultatory signs that supplement data obtained by other methods of examination Very little happens to a heart that does not give some auscultatory clue to the trained observer

A more recent upsurge in interest in cardiac auscultation has resulted from phonocardiography and cardiac surgery Marked improvement in instruments for recording heart sounds and murmurs together with improved methods for correlating these with other cardiac phenomena has advanced our knowledge of auscultation The advent of surgery of congenital and acquired heart disease has led to

important advances in our knowledge of auscultation The ability to correlate what is heard with the pathologic findings at an operation instead of at autopsy has been of inestimable value The knowledge thus obtained has made cardiac auscultation the single most important guide to surgery of acquired valvular disease

The purpose of this book is to indicate what constitutes skillful auscultation and to describe the normal and abnormal auscultatory findings insofar as the trained ear can recognize them Fortunately the trained ear is probably still the best instrument for recognition of most murmurs In the field of the heart sounds extra sounds and gallop rhythms phonocardiography often gives information not available to the ear however an attempt will be made to describe only what the ear can recognize Theory will be given only insofar as it makes more understandable the auscultatory findings The information contained in this book represents the work of numerous people absorbed and digested by the author over many years with few exceptions however I have made no attempt to give the credit that is due I fully realize my debt to these individuals and gratefully acknowledge their help

For help in the preparation of this book I wish to thank the following Drs A A Luisada A Lanari S G Blount M Wassermil C R Hawes and A J Stone for reading the manuscript and offering many valuable suggestions Marjorie Kimmerle for checking the grammar Glenn Mills and William Wheeler for the photography Ruth Kantor for many of the illustrations Jan Ellzey for typing the manuscript and Dr Rose Ravin my wife for everything

A R

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CHAPTER 1

Sound

SOME PROPERTIES OF SOUND

SOME CONCEPTION OF the production characteristics and trans mission of sound is essential for the intelligent discussion of heart sounds and murmurs Only features pertinent to the present discus sion will be covered

Vibrations of a certain frequency range and intensity when they reach the ear give the impression of sound Sounds differ from one another in three respects *pitch quality* and *loudness*

PITCH —The frequency of the vibration determines the pitch of the sound and the lower the frequency the lower the pitch If the vibrations are less frequent than 20 per second or more than 20 000 per second they cannot be heard by the human ear The pitch of many of the sounds emitted by the heart is in the lower range of human audibility thus 80% of the energy of the first and second heart sounds is in frequencies below 70 vibrations per second and most sounds and murmurs are composed of frequencies below 500 per second Frequency components over 650 are of little impor tance in auscultation People differ in their capacity to hear sounds at the upper and lower limits of audibility and the ability to hear low frequency vibrations is a great advantage This may possibly be improved by training

QUALITY —In addition to a fundamental vibration which deter- mines the pitch most sounds have higher frequency vibrations called overtones which determine the quality of the sound Thus the same note has different overtones when produced by a piano violin or trumpet the resulting difference in quality permits one

to recognize the note as coming from the respective instrument

LOUDNESS AND INTENSITY —Intensity in a strict sense refers to the physical aspect of the sound whereas loudness refers to the subjective aspect The intensity of a sound is proportional to the amplitude of the vibration and is independent of the ear Loudness corresponds to the degree of sensation produced and is dependent both on the intensity of the sound and on the sensitiveness of the ear to that particular sound The ear is most sensitive to sounds in the frequency range between 500 and 5 000 Below a frequency of 500 the sensitivity of the ear decreases rapidly A sound with a frequency of 500 vibrations per second will be louder to the ear than a sound of 100 vibrations per second even if the two sounds have the same intensity Since many of the sounds encountered in auscultation have frequencies below 100 vibrations per second and most sounds are below 500 the ear is unfortunately at its poorest in the range where the main portion of the heart sounds and murmurs occur

Although loudness and intensity are thus in a strict sense not the same in general and throughout this book the term intensity is used in the same sense as loudness

Associated with intensity is the phenomenon of masking —a reduction in the ability of the ear to hear certain sounds in the presence of other sounds The ear adjusts itself to the intensity of the sound that it is receiving A loud sound causes the ear to protect itself by cutting down on its ability to receive sound If a faint sound follows immediately on a loud sound the ear is not adjusted to hear it Because of this phenomenon faint murmurs that follow loud sounds are heard with difficulty or not at all The same is true of faint sounds that follow loud murmurs

Masking of a different type occurs when a complex sound—one consisting of several tones of different frequencies—undergoes an increase or decrease of intensity At any given level of intensity masking of some of the tones by others is likely to occur The masking effects will however vary with intensity With changes in intensity some tones may become masked or unmasked and thus change the quality of the sound When a murmur is transmitted to a different area of the chest and the intensity diminishes the quality of the sound may change sufficiently to raise some doubt as to whether the murmur is actually a transmitted murmur

In addition to pitch quality and loudness sounds have *duration* in that they may be short or long The spacing of sounds in relationship to other sounds gives rise to *timing*

TRANSMISSION OF SOUND

Sounds produced in and about the heart are heard only after transmission to the chest wall and then to the ear Some of the factors involved in the transmission of sound are as follows

1 As sound leaves the source the intensity diminishes with the square of the distance from the source Sounds are loudest usually in the part of the chest that is closest to the point at which the sound is produced

2 As sound is transmitted to the chest wall it is influenced by reflections that take place at changes in media i e from heart to surrounding muscles to chest wall or lung etc When sound passes from one medium to another part of the sound is reflected and part passes through Various factors influence the amount of reflection but difference in density of the tissues at the interphase is most important If the media are of approximately the same density most of the sound passes on and only a small part is reflected if the media are markedly different in density a large portion of the sound is reflected Tissues such as blood and muscle are of about the same density and the sound passes through without much reflection On the other hand the lungs with their air spaces have a lower density and sound in going from muscle to lung and from lung to the chest wall suffers a good deal of reflection and is not well transmitted Because there is such a difference in the density of the chest wall and of air only the very loudest of sounds can be heard unaided outside the chest

3 When sound passes through a medium there is a loss of intensity due to friction within the medium This may not affect all frequencies equally therefore the sound in addition to losing intensity may undergo a change in quality since quality depends on the relative frequency make up of that sound Thus as one follows the same murmur over the chest the quality may change This change and the change in quality due to masking must be remembered in deciding whether there are one or two murmurs on the basis of the quality of the murmur heard in different areas

CHAPTER 2

The Stethoscope

DESCRIPTION

FOR TRANSMISSION OF the sounds from the chest wall to the ear the binaural stethoscope is the most commonly used and the observations described in this book have been made with this stethoscope Various types of stethoscopes are used and a basic knowledge of the properties of each is essential Since stethoscopes have a certain individuality it is wise to become familiar with one type and use it most of the time

EAR PIECES —For the transmission of sound through a stethoscope the system should be airtight any leak markedly attenuates the sounds It is therefore important that the ear pieces be of the right size and shape and that they fit the ear well the axis of the ear piece should be parallel to the long axis of the external auditory canal Enough tension must be present in the spring to hold the ear pieces tightly in place

RUBBER TUBING —The shorter the rubber tubing the more efficient the stethoscope Increasing the length of the tubing decreases the efficiency of the stethoscope in transmitting higher frequencies (over 100 cycles per second) The tube length should therefore be a compromise between efficiency and convenience in use A tube length of 10 to 12 inches is usually a good compromise A fairly thick walled rubber tubing is more efficient than a thinner more flexible tubing An internal diameter of ⅛ inch has been shown to be more efficient than the usually used ³⁄₁₆ inch tubing

CHEST PIECES —There are two basic types of chest pieces the bell type and the diaphragm type Bell chest pieces (Fig 1 *A*) are

of different sizes and shapes *The larger the diameter of the bell the* more efficiently it transmits low pitched sounds However there is difficulty in placing the larger bells on the chests of thin patients and children The 1 inch diameter has been most commonly accepted

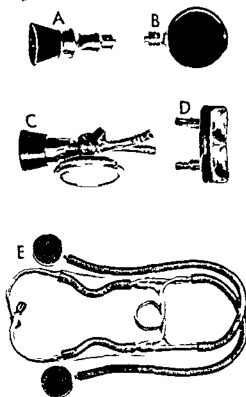


FIG 1 —Stethoscope chest pieces (See text) *A* bell chest piece *B* diaphragm chest piece (Bowles type) *C* Sprague Bowles chest piece *D* Hartert stereostethophone *E* symballophone (Kerr)

as of sufficient size and still small enough for good placement The internal configuration of the bell should be one which keeps the volume as low as possible but does not easily fill with skin and thus decrease the effective diameter The bell chest piece if used correctly (p 19) is best for low pitched sounds

The diaphragm chest piece (Bowles type) (Fig 1 *B*) has a rigid bakelite diaphragm The size varies but a diameter of 1¾ inches

is most commonly used The diaphragm has a relatively high natural frequency and improves the response to higher frequencies but it does this at an over all loss of sensitivity which is especially noticeable in the lower frequencies The diaphragm chest piece is valuable for high pitched murmurs and sounds and helps to overcome the masking effect of loud heart sounds on faint high pitched murmurs that follow the sounds A loss in intensity due to the rigidity of the diaphragm is partially compensated by the increased opening of the diaphragm as compared with the bell Both types of chest pieces should be used in listening to most hearts the open bells for general listening and for the lower frequencies the diaphragm for higher frequencies Because the diaphragm and the bell are always available in the Sprague Bowles type of chest piece (Fig 1 *C*) many have preferred this instrument

Differential or stereostethoscopes —These are of value for localizing and comparing sounds The symballophone (Kerr) (*Fig 1 E*) *has two chest pieces each connected to both* ears but with a longer tubing to the ear opposite the respective chest piece This stethoscope is of value in the following circumstances (1) comparing the intensity of a murmur or sound in different areas This use is of special value in determining the location of the point of maximum intensity of a murmur (2) timing of murmurs or sounds This is done by placing one chest piece in an area where the first or second sound is clearly heard and recognized and the other chest piece in an area where an unusual sound or murmur is heard that cannot be timed (3) distinguishing small differences in pitch and intensity of heart sounds and murmurs

The Hartert stereostethophone (Fig 1 *D*) has two cups close together with the sounds from one cup going to one ear and the sounds from the other cup going to the other ear This very simple chest piece is of special value in determining the location of maximum intensity of a murmur

Amplifying stethoscopes —These are of definite value to some doctors and for some patients They are of value to persons whose hearing is deficient but they should not be used as a substitute for a quiet room Most murmurs of significance can be heard without any amplification Amplifying stethoscopes with filters are of value for training oneself and for teaching In any system with amplification the sound may be different from that ordinarily heard with the binaural stethoscope The examiner must understand his

instrument and be able to recognize the artifacts that are inherent in the instrument and those that result from technical and me chanical difficulties

PHONOCARDIOGRAPHS —The production of more adequate in struments for the recording of heart sounds and murmurs and a bet ter understanding of the physical principles involved have opened up a new field of investigative work With these instruments sev eral advantages are evident (1) the full spectrum of frequencies can be recorded and a permanent record can be obtained (2) am plification permits a better study of faint sounds (3) recording other cardiac events at the same time permits the timing of sounds and murmurs with accuracy (4) by the use of filters vibrations of se lected frequency ranges can be recorded Such recording permits a study of the frequency make up of murmurs By the amplifying of selected bands of frequencies (selective phonocardiography) certain murmurs can be made more evident

Although phonocardiography is an excellent means for increasing one s personal knowledge and for doing investigative work it is really not part of everyday auscultation Its greatest clinical value may be in congenital heart disease

A record of heart sounds is only as good as the person who takes it To have the technician put a microphone in a stated heart area and take a record is essentially worthless The recordings vary with (1) small changes in location (2) amplification (3) back ground noise in the machine room and patient (4) vagaries of the recording instrument and the skill with which the instrument is used (5) the type of chest piece (6) structural factors in the patient

Spectral phonocardiography a new technique differs from con ventional phonocardiography principally in that the frequency spec trum of the sounds and murmurs is more evident This permits a better definition of quality

USE OF THE STETHOSCOPE

QUIET BACKGROUND —Much of what is wrong with everyday auscultation results from its being hurriedly done in a noisy room For satisfactory auscultation the room *must* be quiet One often has the experience of hearing a faint murmur one day and not the next A common cause is the variation of level of background noise When one person is listening other members of a group must be quiet The patient should be comfortable and warm to avoid muscu

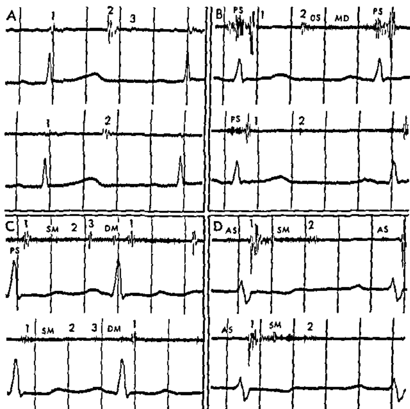


FIG 2 —Effect on heart sounds and murmurs of pressure on bell chest piece In each case the upper tracing was taken with the bell chest piece held lightly and the lower tracing with the bell chest piece applied with pressure

A normal child With light pressure all three heart sounds are clearly heard With pressure the first sound becomes very faint the second sound is less inten e but well heard and the third sound is not heard

B patient with mitral stenosis The upper tracing shows a loud presystolic murmur (*PS*) and loud first heart sound The second sound is followed by a faint opening snap (*OS*) and a middiastolic murmur (*MD*) The rate is slow and the middiastolic murmur begins to fade before auricular contraction pro duces a loud presystolic murmur With pressure on the bell chest piece (low er tracing) the presystolic murmur which in this case is a rather harsh murmur continues to be well heard although it is less intense The first and second heart sounds are diminished in intensity No middiastolic murmur is heard In nearly all patients with mitral stenosis a middiastolic murmur is present but because of its low pitch it may not be heard if the stethoscope is applied with too much pressure The presystolic murmur which is more harsh continues to be heard

C patient with a marked mitral insufficiency Upper tracing shows a sys tolic murmur (*SM*) persi ting throughout systole a rather faint second sound and a well heard third heart sound Because of the rapid rate the rumbling diastolic murmur (*DM*) is essentially presystolic Lower tracing

lar noises Frequently respiration of the patient must be suspended while one is listening

POSITION OF THE PATIENT —For a thorough examination auscultation must be done with the patient in a sitting lying and left lateral recumbent position An adequate examination can possibly be done in the recumbent position alone but listening to the heart with the patient only in a sitting position is absolutely inadequate Typical diastolic murmurs of good intensity when the patient is lying down may not be heard at all when the patient is sitting In the recumbent position the patient s arms must not be held over the head since this will elevate the rib cage and decrease the intensity of the heart sound Positions most commonly of value for different murmurs will be indicated later

APPLICATION OF THE CHEST PIECE TO THE CHEST WALL —Although every doctor uses a stethoscope very few have been taught or have learned by themselves that in use of the bell chest piece there is a marked variation in what is heard depending upon whether the bell is applied very lightly or heavily to the chest wall When the bell is applied very lightly low pitched sounds are well heard Such sounds are the heart sounds especially the first and third many gallop sounds and the middiastolic murmur of mitral stenosis With heavy application of the bell the loudness of these sounds is markedly diminished in most individuals (Fig 2) especially if the amount of subcutaneous tissue is abundant The difference in loudness of middiastolic murmurs and third heart sounds (Fig 2 *A* and *B*) is of a degree that involves the ability to hear or not to hear these sounds Third heart sounds are infrequently heard in part because the pressure of the bell on the skin is too great

This phenomenon has been explained (Rappaport and Sprague*) as follows When the bell is applied to the skin the en

* Rappaport M B and Sprague H B Physiologic and physical laws that govern auscultation and their clinical application *Am Heart J* 21 257 318 1941

The systolic murmur as recorded seems somewhat less intense but this is merely because of a decrease in a few of the lower pitched vibrations and actually the systolic murmur to the ear is very little changed The third heart sound and diastolic murmur are markedly diminished

D patient with an atrial septal defect Upper tracing A clear auricular sound (*AS*) is present The first sound is followed by a holosystolic murmur (*SM*) and a split second sound Lower tracing With pressure the auricular sound can no longer be heard although the phonocardiogram shows a few small vibrations The systolic murmur is essentially unaffected

closed skin forms a diaphragm With increased pressure the skin diaphragm is made more taut and its natural period of oscillation increases This improves the response to higher pitches but at the same time there is a general lowering of the sensitivity of the skin diaphragm As a result the lower frequency components of the heart sounds are attenuated whereas the higher frequencies are still well heard

This important maneuver of being able to diminish the intensity of low pitched sounds by pressure on the bell has several implica tions and applications

1 To hear faint low pitched sounds the examiner *must* hold the bell lightly on the chest wall

2 By noting what happens to a sound or murmur when the bell is first held lightly and then with pressure the examiner can judge to a certain degree the pitch of a sound or murmur

3 The high pitched systolic murmur of mitral insufficiency is less likely to be affected by pressure than medium pitched innocent sys tolic murmurs

4 With pressure the faint high pitched systolic murmur of mi tral insufficiency is less affected than the first heart sound and the masking effect of the first heart sound is thus diminished The same is true of the high pitched early diastolic murmur of aortic insuffi ciency and an accentuated second heart sound

5 In some very noisy hearts with both systolic and diastolic mur murs it is difficult because of the amount of sound in systole to be sure if there is a low pitched diastolic murmur with pressure the total sound is diminished and the low pitched murmur in diastole may disappear By holding the bell alternately lightly and heavily and by concentrating on diastole the examiner can detect the di astolic rumble

LOCATIONS ON THE CHEST WALL —Too often auscultation con sists of applying the stethoscope merely to the apex Most murmurs are called apical because the apex is the only area where the stethoscope was placed Whereas auscultation at the apex the fourth left intercostal space and the second right and left intercostal spaces may be sufficient when nothing unusual is heard tracing the point of maximum intensity and transmission of a murmur may require lis tening in numerous areas over the chest and into the neck back lung bases and even elbows

The auscultatory areas associated with the different valves (Fig

19) are fortunately more widely separated than the projection of the valve areas on the frontal plane This circumstance results from factors occurring on transmission of the sound from the valves to the chest wall Sounds produced at the mitral valve are usually best heard at the apex those at the tricuspid valve just to the left of the lower end of the sternum those at the aortic valve in the second intercostal space just to the right of the sternum and those at the pulmonary valve in the second intercostal space just to the left of the sternum (Fig 19)

LISTENING TO THE HEART —It is true that the person proficient in auscultation may often quickly recognize a lesion just as one recognizes a dog by its bark Most doctors are not trained to the point where they can do this and in many hearts because of multiple modifying factors the bark may be well disguised It may be necessary to listen long and repeatedly to understand completely and analyze what is heard

The untrained person must learn to analyze separately the various sounds and phases of the heart cycle Man has the capacity to direct his attention to the sound he wants to hear He can listen to the person talking in front of him or ignore what is being said and listen to distant sounds This ability to direct attention is the keynote to successful auscultation One listens to the first heart sound and then to the second heart sound and then specifically to systole and then to diastole Sounds other than those being listened to are ignored This method permits the ear to hear all that should be heard and partially to overcome masking I have often had the experience after listening intently for a middiastolic murmur for several minutes in various positions of being asked whether there was a systolic murmur and of being unable to answer because I had not been listening to systole One of the greatest aids in learning to direct attention is the graphic method of recording described below

A sense of rhythm helps in auscultation and the examiner often subconsciously beats out a rhythm while listening If a third heart sound is being listened for and the examiner beats out lub dub puh in his mind while listening to the heart he more easily directs his attention to the point of the cycle where the puh should be heard

CHAPTER 3

Graphic Recording of Auscultation

H N SEGALL* in 1933 described a graphic method for recording auscultatory findings which has many advantages over written descriptions (1) the examiner is forced to listen carefully and separate what is heard into its components—a procedure that as has been previously emphasized is the foundation of good auscultation (2) what is heard can be graphically shown in a fraction of the time taken for writing the same data (3) the graphic description is usually much more complete than a written description because one cannot ignore or forget to describe some of the findings A systolic murmur was heard at the apex may suffice for a written description but graphically the intensity and pitch of the murmur must be shown and its relation to the first and second sound indicated (4) a glance serves to show what has been heard and a comparison with previous auscultations can easily be made This graphic method with minor modifications from Segall s original description has been used throughout this book

The heart sounds are represented by rectangular blocks with the height of the block indicating loudness and the width indicating duration of the sound By diagraming the first and second sound in one cycle and the first sound of the next cycle the length of systole and diastole may be shown Thus the sounds at the apex might be indicated as in Figure 3 *A* A third heart sound is shown in Figure 3, *B* The pitch of a sound is not shown but if unusual it may be indicated by a word or phrase A slightly split first heart sound is shown by notching the rectangle (Fig 3 *B*) A more widely split or redupli

* Segall H N A simple method for graphic description of cardiac auscultatory signs Am *Heart J* 8 533 1933

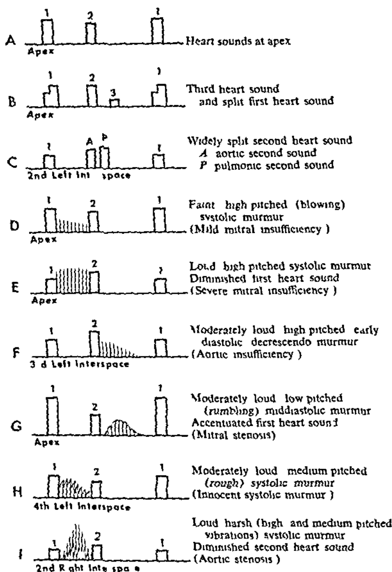


FIG 3 —Symbols used in graphic recording of auscultation

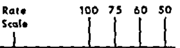




Fig 4—Graphic representation of auscultatory findings The symbols used are described in the text and in Figure 3 The dots indicate the location on the chest wall where the stethoscope is placed The heart rate is approximately 70 per minute this is shown by the distance between

the first heart sounds as compared with the Rate Scale (upper right corner)

The findings at the apex are illustrated for two positions recumbent (circled *R*) and left lateral (circled *LL*) The bell (circled *B*) was used for listening in both positions The first sound is accentuated as indicated by the fact that it is taller than the first sound in Standard for Sounds at Apex (upper left) A faint highpitched systolic murmur is present The number 1 indicates the intensity grade of the murmur (p 64) the intensity grade is not essential but is occasionally helpful Since this murmur was not changed by respiration (in contrast to another systolic murmur) the phrase unchanged by respiration is added on the chart A moderately loud low pitched rumbling middiastolic murmur with a presystolic accentuation is present The number 3 above the murmur is the intensity grade The presystolic portion of the murmur is of higher pitch than the middiastolic murmur (peaked tops instead of rounded tops) The murmur is louder in the left lateral position than in the recumbent position The illustrated auscultatory findings are those of a tight mitral stenosis with a slight mitral insufficiency

Also shown in the diagram is the presence of a faint high pitched systolic murmur localized to a small area at the lower end of the sternum (shaded area) and showing an increased loudness on inspiration This is the murmur of tricuspid insufficiency

The heart sounds in the second right and second left intercostal spaces are shown

cated second heart sound is shown by making two blocks next to each other (Fig 3 *C*)

A high pitched murmur is represented by closely placed vertical lines Timing and duration are shown in relation to the heart sounds The height of the lines indicates the loudness A faint high pitched systolic murmur and a loud high pitched systolic murmur are indicated in Figure 3 *D* and *E* respectively A high pitched early diastolic murmur is shown in Figure 3 *F* A low pitched murmur is indicated by vertical lines with their tops connected by curved lines A low pitched middiastolic murmur is shown in Figure 3 *G* Medium pitched murmurs are indicated by connecting the tops of the vertical lines with peaks thus a moderately loud medium pitched systolic murmur is indicated as in Figure 3 *H* Harsh murmurs are usually loud and contain medium and high pitched vibrations (Fig 3 *I*)

The location on the chest at which the observations are made is recorded on a diagram of the chest with the heart outline and thoracic cage shown (Fig 4) The horizontal lines on the right and left are connected by lines to the area on the chest where the stethoscope is placed For a guide in determining the size of the blocks used to indicate sounds a standard for sounds at the apex is shown on the diagram (upper left) To facilitate determination of the length of the cardiac cycle a rate scale is shown (upper right) The first sound of the cycle to be described is placed at the marker on the line that corresponds to the beginning of the rate scale and the first sound of the next cycle is placed under the number corresponding to the heart rate Careful attention to this phase of the description is not usually necessary but is helpful when one is first learning to use this method The chest piece used in auscultation is indicated by circling the *B* (bell) or *D* (diaphragm) and the position of the patient by circling the *S* (sitting) *R* (recumbent) or *LL* (left lateral recumbent)

To make comparisons more exact one may at times place along with the murmurs the loudness number (p 64) Thus in Figure 4 are represented at the apex an accentuated first sound a very faint (grade 1) high pitched systolic murmur and a moderately loud (grade 3) low pitched diastolic murmur (middiastolic and presystolic) The bell chest piece was used and the findings are shown with the patient in both the recumbent and left lateral positions

The point of maximum intensity of a murmur may be indicated by

an X on the diagram and the area in which a murmur is heard may be shaded

If the examiner knows for example that the first sound must be shown he listens to see if it is normal accentuated or diminished or split The same is true for other sounds If a murmur is present and shown in correct relation to the sounds there can be no question as to whether it is systolic or diastolic Time and again a student will listen to a systolic murmur and say it is diastolic or vice versa If he is asked to listen to the first sound and represent it and then to the second sound and represent it the murmur is almost always correctly placed

The tendency to careless listening and even more careless recording is avoided by this method The method need not be used by all doctors on all patients but it would be well for cardiologists to use it most of the time and most doctors to use it some of the time

CHAPTER 4

Heart Sounds

TIMING OF HEART SOUNDS

1 If the time relationship pitch and intensity are characteristic the first and second heart sounds are usually recognized with no dif ficulty however at rapid rates systole and diastole become almost equal in length (systole equals diastole at a rate of about 120), and the pitch and intensity of heart sounds may be widely altered Some times when a tachycardia is the cause of the confusion the heart may be temporarily slowed by having the patient hold a deep breath or by pressure on the carotid sinus

2 The first sound occurs at the onset of the apical impulse and the carotid pulsation The sounds should *not* be timed by the radial pulse which is just late enough to be confusing The apical impulse is probably better than the carotid pulsation in most people If the stethoscope or hand is put at the apex the first heart sound will be synchronous with the outward thrust

3 Gradually moving the stethoscope from an area where the sounds are clear and timing definite to areas where timing is not clear will sometimes be of value

4 The use of the symballophone is often helpful if one chest piece is kept on the area where the sounds are clearly recognized and the other chest piece on the area where they cannot be recog nized

5 The graphic recording of the heart sounds along with an elec trocardiogram or jugular pulse tracings is sometimes necessary for timing

FIRST HEART SOUND

Description

Although several cardiac events play a part in the production of the first heart sound the closure of the atrioventricular valves is the most important and accounts for most of the sound that is heard Phonocardiographic analysis (Fig 5) shows four components in the first sound which have been related to the various events occurring at the onset of systole (1) development of tension in the ventricular musculature (2) closure of the atrioventricular valves (3) opening of the semilunar valves and the onset of ventricular ejection and (4) acceleration of the blood in the arteries during maximum ejection Often some residual vibrations of auricular origin occur at the very beginning of the first sound Normally only the components due to the closure of the AV valves and the opening of the similar valves are heard but the other components may be heard under abnormal circumstances

Since the different components of the first sound have varied frequencies the first sound cannot be accurately classified as having a definite fundamental vibration there is rather a group of vibrations most of which belong in the 40 to 60 per second range As a group these vibrations are only slightly lower in pitch than those of the second sound but the second sound has more high pitched overtones If therefore the bell chest piece is pressed firmly to cut down on low pitched vibrations the intensity of the first sound is decreased more than that of the second sound

The first heart sound is normally loudest at the apex In this region it is usually louder than the second heart sound but the two sounds may be of about equal loudness and the second sound may at times be louder than the first—especially if the bell chest piece is applied too firmly At the base of the heart the first heart sound is usually louder in the second left interspace than in the second right interspace and in both areas the second heart sound is louder than the first

Changes in loudness of the first sound and the presence and degree of splitting are noted on auscultation Variations in pitch are often associated with changes in loudness When the pitch is higher than usual the sound is usually loud and sharp low pitched sounds are dull or muffled When the sounds are short sharp and rapid they leave an impression that has been described as tick

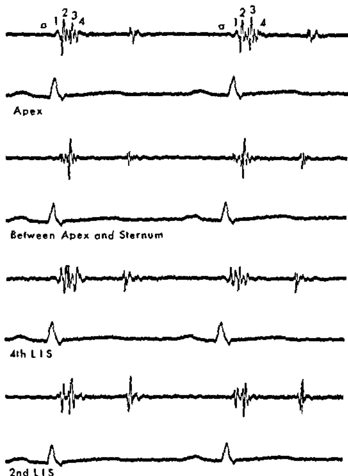


FIG 5 —Variation in first heart sound in different locations In the tracing at the apex the four components of a first heart sound that can be recognized on a phonocardiogram are indicated The first and fourth components are not usually heard An auricular sound (*a*) is often evident on the phono cardiogram but not usually heard in normal persons The variation in the components in different beats is produced by respiration

The second and third components can often be distinguished *on ausculta tion* especially when as clearly separated as in this patient The first sound then consists of two components usually the first component (actually the second component as noted above) is loudest at the apex and the second component is loudest along the left border of the sternum (see text) Even when the two components are as clearly separated in most areas as they are in this patient a blending occurs in some areas (*4th L I S*) that suggests that the first heart sound consists of more than a combination of two clear cut components such as is seen in the case of the second heart sound

tock The quality of the first sound is less significant than the loud ness or pitch although occasionally the sound may have an unusual quality—for example the resonating and tonelike first sound some times heard in mitral stenosis

Changes in Loudness

The factors that influence the loudness of the first sound may be *extracardiac* or *cardiac* The extracardiac factors usually affect both the first and the second heart sounds Cardiac factors involve pri marily the first sound

Extracardiac Factors Influencing the Intensity of the First Heart Sound

1 Thickness of the chest wall —The farther the sound has to travel through the chest wall the fainter the sound will be because of distance dampening and reflection In a heavy chested person the sounds will be less intense than in a thin-chested person or in children Fatty tissue is inelastic and a poor transmitter of sound and therefore the sound will be faint in obese persons and women with large breasts Sounds will be less evident in a patient with a deep chest that is a person with a large anterior posterior chest di ameter In these patients the sounds are better heard in the sitting than in the recumbent position

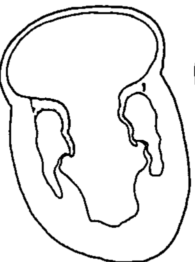
2 Emphysema —Emphysematous lungs can effectively insu late the heart so that the heart sounds will be faint or inaudible

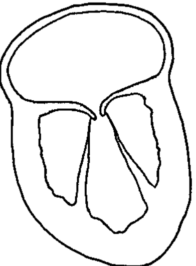
3 Pericardial fluid —This may diminish heart sounds both by increasing the distance from the heart to the chest wall and by in troducing new reflections Many patients with myxedema have peri cardial fluid that may account in part for a decreased intensity of the heart sounds

Cardiac Factors Influencing the Intensity of the First Heart Sound

The cardiac factors consist of (1) position of the atrioventric ular valves at the time of ventricular contraction (2) valve struc ture (3) force and abruptness of ventricular contraction (4) mask ing

1 Position of the atrioventricular valves at the time of ventricular contraction —Variations in intensity of the first sound have been closely correlated with the position of the atrio





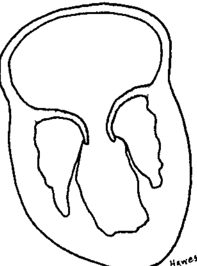


FIG 6 —Relation of position of atrioventricular valves at time of ventricular contraction to intensity of first heart sound

ventricular valves at the moment of ventricular contraction If the valves are widely open when the tension in the ventricle is increased they snap shut with production of a loud sound If valves have fallen back to the position of closure and are thus partially taut the amount of sound produced at the time of ventricular contraction is greatly diminished The amount of sound produced by closure of the valves from intermediate positions will vary accordingly (Fig 6)

The position of the valves during diastole is influenced by the flow of the blood from the auricles to the ventricles and by auricular contraction Immediately after the opening of the valves there is a period of rapid blood flow during which the valves are wide open As the flow slows after this sudden filling the valves are partially taut The valves then open again but not as widely since the flow is slower With auricular contraction the valves are again wide open after which they fall toward each other again Since the intensity of the first heart sound varies with the position of the atrioventricular valves at the time of ventricular contraction the intensity depends on the time in diastole at which ventricular contraction occurs (Fig 6)

Variations in loudness of the first sound in a number of clinical conditions have been closely correlated with the expected position of the atrioventricular valves at the time of ventricular contraction

a) Complete heart block—The pathognomonic variation in the intensity of the first heart sound in complete heart block has been shown to depend upon the varying relationship of auricular systole to ventricular systole (Fig 7) In patients with this condition the first heart sound is most accentuated when the P wave precedes the QRS wave by 0 08 second to 0 12 second and the valves are wide open The loudness of the first sound decreases as the P R interval lengthens and with P R intervals of over 0 20 second and the sound may be markedly diminished By this time the valves are partially closed In some patients there may be a secondary zone of accentuation of the first sound when the P R interval increases over 0 25 second Since a bradycardia with a constant P R interval (sinus bradycardia or 2 to 1 block) will not show this variation in the first sound this sign is a valuable diagnostic aid

b) Acute rheumatic fever—In acute rheumatic fever there is often a prolongation of atrioventricular conduction Accompanying this change in the P R interval is a change in the loudness of the

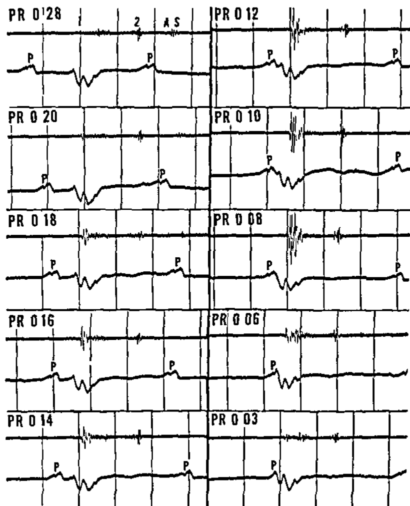


FIG 7 —Variation in intensity of first heart sound in complete heart block When the P wave precedes the QRS complex (W shaped in this patient) by 0 20 second or over (upper left) the first sound is very faint The first sound is somewhat louder when the P R interval is 0 18 second When the P R interval is 0 16 and 0 14 second the first sound is moderately loud At 0 12 second and 0 10 second the first heart sound is loud and at 0 08 second it is very loud It drops off sharply at 0 06 second and becomes very faint at 0 03 second

In the upper left strip (PR 0 28) note that the auricular contraction (*A.S.*) produces a very audible sound when it occurs in early diastole and is superimposed upon the phase of rapid ventricular filling At other times the auricular sound was sometimes evident but faint

The time interval between the vertical lines in all the phonocardiograms shown in the text is 0 2 second

first heart sound When the P R interval is 0 12 second the first sound is sharp and louder than the second sound At 0 14 second the first sound is moderately sharp and louder than the second sound at 0 16 second the first and second sounds are of about equal intensity at 0 18 second the second sound is louder than the first sound and the first sound is definitely diminished in intensity at 0 20 second the first sound is faint Since this correlation between P R interval and intensity of the first sound is good one has a simple clue to the presence of prolongation of atrioventricular conduction in rheumatic myocarditis It is to be noted that the decreased loudness of the first heart sound is a function of the P R interval and is not produced, as has often been considered in the past by myocardial weakness Even in the absence of rheumatic fever a definitely diminished first heart sound as compared with the second heart sound at the apex should make one suspect the presence of a P R interval at the upper limit of normal or prolonged

c) Ventricular tachycardia —In some instances of ventricular tachycardia in which the auricles are beating at an independent and slower rate than the ventricles the ventricular beats that follow very shortly after auricular beats have an accentuated first sound An accentuated first sound thus occurs at fairly regular intervals The explanation is similar to what has been previously noted The variation in first sound does not occur in the presence of auricular fibrillation or if there is retrograde conduction to the auricles This sign may be of value in distinguishing ventricular tachycardia from other types of tachycardia

d) Auricular fibrillation —In auricular fibrillation the first sound varies in loudness with the length of the preceding diastole in a manner that would be expected from the description given Phonocardiographic studies show that the first sound is loudest when it occurs up to about 0 21 second after the preceding second sound when the valves are wide open during rapid ventricular filling The sound is markedly diminished during the next tenth of a second as the valves fall toward each other at the end of the rapid filling A secondary zone of moderate accentuation usually follows The variation in loudness may be evident to the listener and is of some diagnostic value

e) Mitral stenosis and auricular fibrillation —An interesting relationship between intensity of the first heart sound and the length of the preceding diastole is found in some patients with mitral steno-

sis and auricular fibrillation The first heart sound is loudest when it occurs shortly after the second sound and then gradually diminishes in intensity as the interval after the second sound increases This has been explained as follows Because of the stenosis the ventricle fills slowly Early in diastole therefore the ventricles do not have much blood and since the pressure in the auricles is high the valve is pushed toward the ventricle As the ventricles gradually fill the pressure on the two sides equalizes and the valve gradually rises toward the auricle This would account for a loud sound early in diastole that gradually decreases as diastole lengthens Patients who show this variation in the first sound usually have moderately tight rather than very tight mitral stenosis often with some insufficiency

2 PATHOLOGIC CHANGES IN THE VALVES AS A CAUSE OF CHANGES IN LOUDNESS OF THE FIRST SOUND —When as a result of rheumatic fever the mitral valve is markedly fibrosed calcified and bound down so that there can be little motion the intensity of the first sound is usually diminished

In the presence of mitral stenosis the first sound is loud and high pitched if the valve cusps retain their mobility Several factors may play a part in producing this accentuated sound (1) because of the stenosis pressure in the left auricle is increased filling of the ventricle is slow and the valve remains deep in the ventricle until ventricular systole occurs (2) the interior cusp of the valve may be thickened and shortened and this tends to produce a higher pitched sound (3) the valve ring may be thickened and thus give more support to the valves and the thickened chordae tendineae may stop the motion of the valve more abruptly than the normal chordae tendineae (4) since the pressure in the left auricle is elevated ventricular pressure must rise to a higher level before the valves close The closing pressure is increased and the valve is closed more abruptly

3 EFFECT OF ABRUPTNESS OF VENTRICULAR CONTRACTIONS ON THE LOUDNESS OF THE FIRST SOUND —The abruptness with which ventricular systole occurs—that is the speed with which the ventricles contract—would seem to be a factor which might affect the first sound *since a more abrupt closure of the valves causes the* production of higher pitched frequencies that would make the sound seem louder to the human ear An abrupt ventricular systole would not necessarily be related to the height of the systolic pressure attained or to the amount of blood ejected A more abrupt

closure of the valves might be a factor in the increase of the first sound heard in exercise thyrotoxicosis fever and mitral stenosis and after certain drugs such as epinephrine A slower and less abrupt closure of the AV valves may be a factor in the diminished first heart sounds heard in terminal states myocardial infarction myxedema and some cases of shock In all of these instances other factors may play a part

4 MASKING OF THE FIRST HEART SOUND —Loud systolic murmurs may mask a first heart sound With training one may learn to separate the sound from the murmur even though the first impression is that the murmur has replaced the sound Often a phonocardiogram will show a first sound when none can be definitely heard with the stethoscope

SPLITTING OF THE FIRST HEART SOUND

Heart sounds are produced by similar events occurring synchronously or almost synchronously in both sides of the heart e g closure of the atrioventricular valves closure of the semilunar valves distention of the aorta and pulmonary artery Splitting of a sound usually carries with it the implication of asynchronism of an event in the two sides of the heart This is essentially true in the case of the second heart sound in which all the sound heard is produced by the closing of the semilunar valves The first heart sound however is a more prolonged sound and the audible portion consists of two components (components 2 and 3 p 29) which can usually be differentiated in the phonocardiogram (Fig 5) As already noted the first component is usually considered as produced by the closing of the atrioventricular valves and the second component by the opening of the semilunar valves and the onset of ventricular ejection These two components commonly blend into a single sound often however the two components can be recognized Since such recognition may be a normal finding and occurs with no prolongation of the first sound the term splitting does not seem especially appropriate but can be accepted on the basis of common usage

Splitting when present often shows a characteristic pattern which can be recognized by listening first at the apex and gradually moving toward the sternum (Fig 5) At the apex or somewhat lateral to the apex the sound is usually single Somewhere between the apex and the sternum two components of about equal inten

sity may be recognized Near the sternum the split consists of a less intense first component followed by a louder somewhat sharper second component Near the sternum the first component often has a crescendo character and occasionally the impression of a presystolic murmur is erroneously obtained especially if there is some tachycardia Splitting is more evident in expiration Although this splitting of the first heart sound is often seen in normal persons I believe it is more evident and more common in older persons in patients with hypertension and in patients with cardiac pathology even in the absence of conduction defects

Because of splitting the first sound in the sternal region may have a scraping quality which is much more evident in the sitting position and on expiration A sound called the xiphosternal crunch has been described and although this term seems to have been used for almost any sound occurring in the region of the xiphoid it is true that when the patient is in the sitting position the quality of a split first heart sound in the xiphoid region may be described as crunching

Since the first component is loudest over the apex and the second component along the left border of the sternum it has been suggested that both components are produced by the atrioventricular valves the first component by the mitral valve and the second component by the tricuspid valve This suggestion implies a commonly occurring asynchronism in the closing of the atrioventricular valves in persons with normal ventricular excitation If both components were produced by the atrioventricular valves *bundle branch block* especially right bundle branch block would be expected to increase the asynchronism in the contraction of the ventricles and produce a widely split first sound From an auscultatory standpoint however the changes in the *first sound* in bundle branch block are disappointing only an occasional patient with bundle branch block shows an unusual degree of splitting The usual absence of clear splitting is also noted on phonocardiograms and favors the concept that the two audible components of the first sound are produced by different types of events

If a semilunar and vascular component forms a normal part of the audible portion of the first heart sound then accentuation of the vascular component in certain pathologic conditions seems likely and some of the so-called *systolic ejection sounds* occasionally heard are the result of an abnormally loud vascular component Delay of

this vascular component would occur when the isometric contraction period was prolonged as in pulmonary or systemic hypertension and might result in a clear separation of this component from the valvular component *Pulmonary early systolic sounds* (Fig 8) are occasionally heard in patients with dilated pulmonary arteries

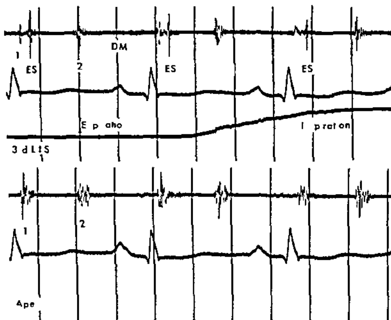


FIG. 8—Pulmonary early systolic sound in pulmonary hypertension with dilation of pulmonary artery This sound (*ES*) is best heard in the second and third left intercostal spaces and is loudest in expiration It has a clicking quality The auscultatory impression is very similar to that of a split first sound with a loud second component The sound is not heard at the apex (lower tracing) A high pitched diastolic murmur (*DM*) is present (upper tracing) and is produced by a pulmonary insufficiency As is generally true a high pitched murmur is much more evident to the ear than on a tracing

with or without pulmonary hypertension and in pure pulmonary stenosis when mild The impression given may be that of an accentuated first heart sound in the second and third left intercostal spaces or of an early systolic, clicking sound This sound is loudest in expiration and is poorly heard at the apex Occasionally a similar sound may be heard in the aortic region in systemic hypertension mild aortic insufficiency or mild aortic stenosis with a poststenotic

dilation This aortic systolic sound is usually well heard at the apex

Auricular contraction may at times produce sufficient sound to give the impression of a split first sound with separation of the components The conditions favoring this increased sound production by the auricle are described on page 55 Abnormal sounds of undetermined origin may occur in close proximity to the first sound and give the impression of a split first sound

SECOND HEART SOUND

Description

The second heart sound is produced by vibrations initiated by the closure of the aortic and pulmonary semilunar valves These vibrations occur in the semilunar valves and in the adjacent portions of the aorta and pulmonary artery The sound produced by the aortic semilunar valve is usually heard over the entire precordium that produced by the pulmonary valve is normally heard in a much smaller area centering around the second left interspace (Fig 9) In expiration the aortic and pulmonary semilunar valves close almost synchronously and produce a single sound With inspiration systole of the right ventricle is slightly prolonged probably because of increased filling of the right ventricle and the pulmonary valves close later than the aortic valves This results in an inspiratory splitting of the second sound which varies in degree and is commonly most marked in the heart beat at the peak or immediately after the peak of inspiration Splitting will be evident only in the area where the pulmonic as well as the aortic second sound can be heard

The degree of splitting varies in different individuals In children some degree of splitting can almost always be recognized In many adults especially if the second sound is not too well heard because of a heavy chest wall or emphysema no splitting can be recognized Often even when a clear splitting may not be evident a definite change in the quality of the sound occurs with respiration A slight degree of splitting may normally be present in expiration The splitting is best determined during ordinary respiration although occasionally if respiration is shallow the patient may be asked to breath somewhat deeper Abnormally deep respiration and held respiration may be confusing In held expiration the second sound which is at first single often becomes split although not usually to the degree noted on inspiration With held inspiration the second sound first split becomes single Recognition of splitting is like

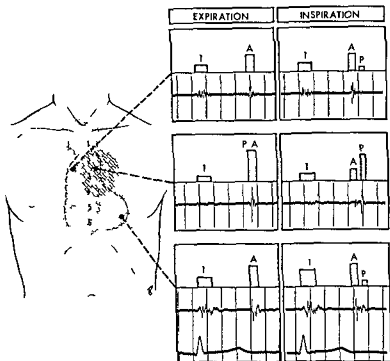


FIG 9—Second heart sound composition in different areas of precordium and effect of respiration The stippling indicates the area where the aortic second sound is heard the shaded portion the area in which the pulmonic second sound is normally heard As is noted the second sound in the second right intercostal space and at the apex is essentially of aortic origin In the second left intercostal space the second heart sound is composed of both aortic and pulmonic second sounds In different persons the relative intensities of the two components in this area will vary and often the aortic component is louder than the pulmonic component even in this area In expiration the aortic and pulmonic components in most normal persons are superimposed and produce a single sound In inspiration right ventricular systole is prolonged and the pulmonic second sound is delayed In the area therefore in which the pulmonic second second is evident a splitting of the second sound will occur In the case illustrated the pulmonic component of the second sound can be seen on the phonocardiogram during expiration in both aortic and apical regions It was not evident however to the ear

everything else a matter of training and direction of attention once the examiner becomes conscious of the occurrence of splitting it opens a new field of auscultatory enjoyment

Phonocardiograms indicate that the pulmonic component of the second sound may be present in the aortic area (Fig 9) however here it is faint masked by a preceding louder aortic component *and not normally heard The aortic second sound is often louder* than the pulmonic second sound in the pulmonic area Both the aortic and the pulmonic second sound may occasionally be louder in the third left intercostal space than in any other area Although the phonocardiogram may show a faint pulmonic sound at the apex the second sound at the apex is almost entirely aortic in origin

Since the second heart sound is usually split in inspiration the expression normally split is used to indicate this finding Splitting of clear degree in expiration is usually abnormal

The wider distribution of the sound produced by closure of the aortic semilunar valves probably results from the much higher closing pressure in the aorta and the deeper location of the aorta The sound produced by the closure of the pulmonary valves is less intense because of the lower closing pressure But since the pulmonary artery is close to the chest wall the sound is well heard though in a small area During the first three to four decades of life the loudness of the second sound in the second left intercostal space is usually louder than that of the sound in the second right intercostal space In older people the situation is reversed This change in relative intensities may result from the increase in the systemic pressure the change in the relative position of the aorta and pulmonary artery and the changes in the structure of the aorta and aortic valves that occur with age

The second sound is normally louder at the base than at the apex At the base the second heart sound is louder than the first heart sound At the apex the second heart sound is not usually as loud as the first heart sound but may be of equal intensity and not infrequently is louder than the first sound

Variations to be noted in the second sound are *changes in loudness* and *changes in degree of splitting* Changes in quality of the second sound occur and sometimes are significant but generally are less important than changes in loudness

CHANGES IN LOUDNESS OF THE SECOND HEART SOUND

An increased or decreased intensity of the aortic second sound results in a change of intensity of the second sound in the second right intercostal space and apex In persons in whom the heart sounds are faint at the base because of emphysema or a heavy chest wall the character of the second heart sound at the apex gives a clue to changes in the aortic second sound for example the second sound at the apex may be increased in hypertension An increase in the pulmonic second sound results in a loud pulmonic second sound in the second left intercostal space Since the sound is also more widely heard splitting of the second sound may be evident in a wide area including the second right intercostal space down the left border of the sternum and often at the apex

The factors that produce changes in loudness of the second sound may be divided into *extracardiac* and *cardiac* The *extracardiac factors* are similar to those that influence the first heart sound (p 31) and the two heart sounds are usually affected at the same time Fibrosis of one or the other of the upper lobes of the lungs may increase the second heart sound in the area without especially affecting the first heart sound

The *cardiac factors* affecting the loudness of the second sound are (1) the pressure in the aorta or pulmonary artery (2) pathologic changes in the valves (3) changes in the structure of the valve ring and great vessels (4) masking

1 ARTERIAL PRESSURE —The greater the closing pressure in the vessel the louder the sound produced by closure of the valve Exercise excitement and essential hypertension increase the pressure in the aorta and may increase the aortic second sound Any of the many conditions associated with pulmonary hypertension—congenital heart disease mitral stenosis congestive heart failure and idiopathic pulmonary hypertension—will produce an increased pulmonic second sound

The faster the pressure falls during the latter part of systole the more abrupt the closure of the valve The result may be a greater proportion of high pitched vibrations and a louder sound The increased aortic second sound in some patients with aortic insufficiency may in part be produced by this mechanism

A fall in systemic blood pressure due to hypotension circulatory failure or shock will result in a diminished aortic second sound With infundibular and valvular pulmonary stenosis the pulmonic

second sound will be diminished or absent owing in part to the low closing pressure

In congestive failure in older patients the relative loudness of the aortic and pulmonic second sounds may show changes that are of diagnostic value When the failure in these patients is compensated the aortic second sound is louder than the pulmonic second sound When failure occurs the pulmonary artery pressure increases and the pulmonic second sound increases At the same time the systemic pressure may decrease and the aortic second sound decrease Thus the pulmonic second sound will be louder than the aortic sound As the patient improves with treatment the pulmonary artery pressure decreases and the systemic pressure increases The aortic second sound first becomes equal to the pulmonic second sound and then becomes louder

2 Pathologic changes in the valves —If the valve remains flexible mild thickening of the valve such as occurs with rheumatic fever and possibly arteriosclerosis may increase the second sound If the valves are markedly thickened and calcified and the motion is limited the second sound is diminished or absent as in aortic stenosis

3 Changes in the structure of the valve ring and great vessels —Arteriosclerotic or syphilitic changes in the vessel may increase sound production Dilation of a vessel may change the quality of a sound and in aortic aneurysm the second sound may have a peculiarly resonant quality Arteriosclerosis of the aorta may increase the aortic second sound especially if there is some dilation Temporary mild hypertension such as occurs in persons who are blood pressure hyperreactors is less likely to be associated with an accentuated second sound than a persistent hypertension of the same degree with aortic changes

4 Masking —A loud systolic murmur may mask a normal or somewhat diminished second sound in aortic stenosis pulmonic stenosis patent ductus arteriosus and mitral insufficiency

Split or Reduplicated Second Heart Sounds

As has been noted inspiratory splitting is normal and is usually evident only in a small area centering around the second left intercostal space Splitting of the second sound may not be evident in normal persons It is difficult to recognize splitting in infants and small children with rapid heart rates and rapid respirations If respi

rations are very shallow splitting may be absent When the intensity of the second heart sound is decreased because of emphysema or a heavy chest wall splitting may not be evident In older persons the pulmonic second sound may be faint and only the aortic second sound may be heard In some normal persons no reason may be evident for the absence of recognizable splitting

Splitting is *absent* in some congenital and acquired heart conditions owing to the following causes (1) only one valve may be producing sound In the tetralogy of Fallot (Figs 12 and 37) only

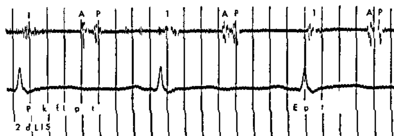


FIG 10—Increased splitting of second sound in right bundle branch block The splitting is evident in expiration and becomes more marked on inspiration The aortic second sound (*A*) precedes the pulmonic second sound (*P*) There is a faint systolic murmur in this patient but most of the vibrations other than the heart sounds are due to respiration and muscle noises

the aortic sound can be heard The second sound may have a pure and tonelike quality (2) one of the sounds may be masked by a loud murmur This masking may occur in aortic or pulmonic stenosis (Figs 36 and 38) (3) one sound may be so accentuated that it masks the other sound This situation may occur with pulmonary or systemic hypertension (Fig 12) By listening in an area where the sound is not so intense one may sometimes recognize splitting e g listening in the fourth left intercostal space in pulmonary hypertension

Splitting may be *increased or altered* in the following conditions

1 RIGHT BUNDLE BRANCH BLOCK—In this condition excitation of the right side of the heart is delayed and as a result the pulmonary valve closure is delayed Splitting of the second sound is present in expiration and is further increased and marked in inspiration (Figs 10 and 12) The diagnosis of right bundle branch block can often be made or suspected on this finding alone

2 LEFT BUNDLE BRANCH BLOCK—Excitation of the left side of

the heart and aortic closure are delayed The aortic component of the second sound will follow the pulmonic component a reversal of the usual state because of this the splitting is more evident in expiration (paradoxical splitting) when right ventricular systole is shortest and the pulmonic second sound early (Figs 11 and 12) The splitting of the second sound in left bundle branch block is not as evident as that in right bundle branch block—possibly because it is more common in age groups where splitting may normally not be evident

3 INCREASED RIGHT VENTRICULAR EMPTYING TIME —In atrial septal defects the increased blood flow through the right ventricle

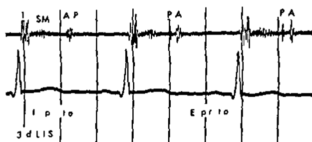


FIG 11 — Paradoxical splitting of second sound in congenital aortic stenosis Because of the aortic stenosis the left ventricular systole is prolonged and the aortic second sound is delayed In expiration therefore when the right ventricular systole is short the pulmonic second sound occurs early and there is splitting In inspiration right ventricular systole is prolonged and the pulmonic second sound now falls at about the same time as the aortic sound giving a single or almost single sound

produces a delay in the pulmonic second sound (Figs 35 and 38) Partial right bundle branch block if present may also play a part With large atrial septal defects the splitting is slightly or not at all increased by inspiration—presumably because filling of the right ventricle is already markedly increased and cannot be further increased by inspiration

Pulmonary valvular and infundibular stenosis produce a delay in the pulmonic second sound if the sound is heard (Figs 12 36 and 38)

4 INCREASED LEFT VENTRICULAR EMPTYING TIME —In most patients with aortic stenosis normal splitting of the second sound occurs if an aortic second sound is present However severe aortic stenosis may be associated with a delay in the aortic second sound because of increased left ventricular emptying time The pulmonic

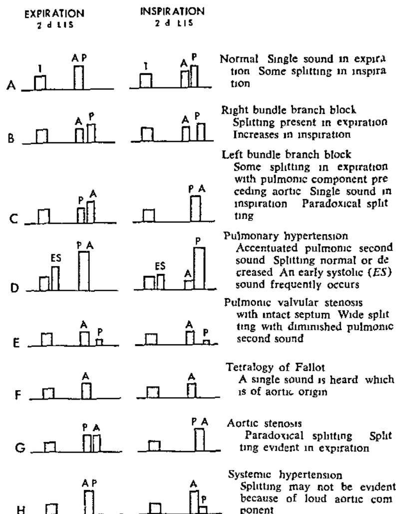


Fig 12—Changes in second sound *A* aortic second sound *P* pulmonic second sound *ES* early systolic sound

second sound then precedes the aortic second sound and a paradoxical splitting occurs since the splitting is most evident in expiration (Fig 11) If a loud systolic murmur masks the pulmonic second sound splitting may not be evident The resulting pure second sound is especially evident in congenital aortic stenosis

THIRD HEART SOUND

The third heart sound occurs shortly after the second heart sound (0 13 to 0 18 second) (Fig 2 A) It is produced either by the closure of the atrioventricular valves at the end of the rapid ventricular filling or by the vibrations of the ventricular walls under the impact of and distention by the incoming stream of blood in rapid ventricular filling Because of its low intensity and low pitch (lower than either of the other heart sounds) it is not commonly heard although it can often be recorded The frequency with which it is heard depends on how often it is looked for in the correct manner (1) it is best heard in children and patients with thin chest walls (2) the room must be quiet and the patient recumbent or in the left lateral position The sound may disappear if the patient sits up (3) the examiner must hold the bell chest piece very lightly and must concentrate on early diastole (4) the third sound is usually best heard at the point of the apex impulse or just medial to the apex impulse (5) it is best heard usually just at the onset of expiration Occasionally a third heart sound produced in the right side of the heart is better heard during inspiration The third sound may disappear if the breath is held after a deep inspiration (6) the sound is increased by exercise pressure on the abdomen or lifting of the legs (7) for some reason the sound is best heard at the very start of auscultation Some type of accommodation occurs with continued listening which makes the sound less evident

If attention is paid to the factors mentioned above a normal third heart sound may be heard in many children and in some adults The occurrence of an abnormally loud third heart sound is discussed under *protodiastolic triple rhythms* on page 58

CHAPTER 5

Abnormal and Extra Heart Sounds

OPENING SNAP OF THE MITRAL VALVE

A STENOTIC MITRAL VALVE that has retained some degree of flexibility may produce a sound when it opens This sound usually occurs 0 08 to 0 10 second after the second sound and has been called the opening snap of the mitral valve Phonocardiograms occasionally show small vibrations at this point in normal persons but usually no audible sound is present In such cases of mitral stenosis the valve resembles a hammock with a hole in the middle (Fig 42 *A*) During systole the hammock bulges into the atrium At the end of systole as the pressure in the left ventricle drops below that in the left atrium the hammock is snapped back and bulges into the left ventricle The higher the pressure in the left atrium the more forcible the movement of the valve and the louder the sound Other factors involved in the increased sound production would be similar to those resulting in an increased first sound in mitral stenosis (p 36)

The sound is high pitched and of a snapping or clicking quality It may be louder in some areas than the second heart sound which it resembles It is always associated with a good and usually accentuated first heart sound The opening snap may be heard over the entire precordium but is best heard along the left border of the sternum at about the fourth intercostal space (Fig 13) In this area the sharp clicking character is marked and the sound can be easily recognized At the apex the other sounds associated with mitral stenosis accentuated first sound and middiastolic murmur may mask the opening click unless attention is directed toward it In the

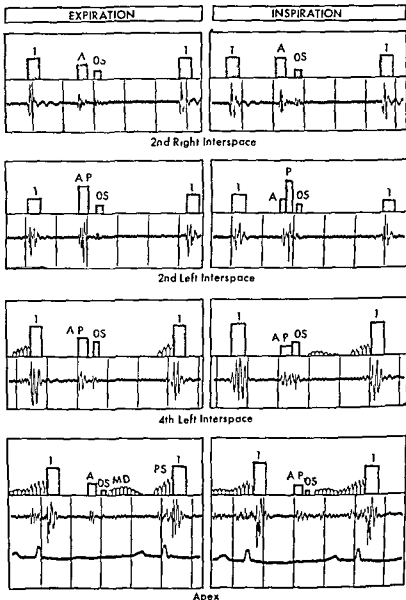


FIG 13 —Opening snap of mitral valve The opening snap of the mitral valve (*OS*) is usually loudest in the fourth left intercostal space In this area it has a clicking sound It is most evident in expiration but is usually recognized in inspiration In the second left interspace the opening snap is usually heard in expiration However in inspiration in this area the pulmonic second sound (*P*) moves into the space between the aortic second

pulmonic area the opening snap must be separated from a split second sound Separation can usually be accomplished by recognition of both the split second sound and the opening snap on the basis of differential changes that occur on respiration these are described in detail below The sound is often well heard in the aortic area where it has less of a snapping quality The opening snap of the mitral valve is probably the most commonly heard extra sound in the aortic area

The opening snap of the mitral valve usually indicates a flexible valve and its presence is evidence that the valve is probably suitable for operation Since it is often an early sign of mitral stenosis the opening snap is not in itself an indication for operation The snap is not heard with few exceptions when the valve is markedly fibrosed and calcified and cannot move—although it is heard at times when the valve is quite thickened It persists after the onset of auricular fibrillation and in most instances after mitral commissurotomy even though the clinical results may be excellent

Since the time interval between the beginning of the second sound and the opening snap of the mitral valve represents the time from the closing of the semilunar valves until the pressure in the left ventricle falls below that in the left atrium the higher the left atrial pressure the shorter will be the time between the second sound and the opening snap Furthermore with increased left atrial pressure the first heart sound will be delayed as the pressure in the left ventricle during isometric contraction builds up to that in the left atrium (Fig 25) If the left atrial pressure decreases as a result of mitral commissurotomy the delay in the first sound (as measured by the interval between the onset of the QRS complex of the electrocardiogram and the beginning of the first heart sound) should decrease and the second sound opening snap interval should increase These changes can be evaluated only on phonocardiograms

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sound (*A*) and the opening snap and it is often difficult to hear the opening snap after the pulmonic sound especially if the pulmonic second sound is accentuated With careful listening all three sounds can sometimes be distinguished The opening snap is usually well heard in the second right intercostal space and is the most common extra sound heard in that area Since the pulmonic second sound is not evident in this area the sound is equally well heard in inspiration and expiration The opening snap is present at the apex but usually is not as loud as in the fourth left intercostal space Often the accentuated first sound and the middiastolic (*MD*) and presystolic murmurs (*PS*) mask the opening snap

but may be helpful as a simple way of evaluating the result of mitral commissurotomy

The opening snap can be distinguished from a split second sound on the following bases

1 A split second sound is loudest in the pulmonic area the opening snap is loudest to the left of the sternum in about the fourth intercostal space When the pulmonic component is accentuated a reduplicated second heart sound may be heard along the left

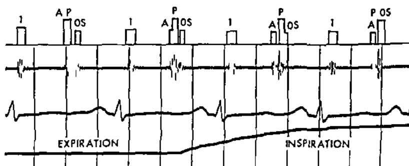


FIG 14.—Opening snap of mitral valve In this patient the opening snap (*OS*) is quite close to the aortic (*A*) and pulmonic (*P*) second sound in expiration and the splitting that occurs on inspiration is enough to place the pulmonic second sound on top of the opening snap The two sounds heard on expiration are therefore (1) the combined aortic and pulmonic second sounds and (2) the opening snap On inspiration the two sounds are (1) the aortic second sound and (2) the combined pulmonic second sound and opening snap

border of the sternum but the splitting is still most evident in the pulmonic area

2 With careful listening in the pulmonic area during respiration both the split second sound and the opening snap can be recognized On expiration the splitting of the second sound is minimal or absent and the opening snap is evident at a definite interval after the second sound (Figs 13 and 14) On inspiration the splitting of the second sound is increased and becomes evident (Figs 13 and 14) The opening snap maintains a constant interval to the aortic or first component of the second sound and when the second sound is split the pulmonic component moves into the interval between the aortic second sound and the opening snap Since the pulmonic second sound is often somewhat accentuated the opening snap which now immediately follows it is heard with difficulty When the

pulmonic second sound is not loud all three sounds may often be recognized in inspiration a very obvious snap that follows a single second sound is heard in expiration When the opening snap closely follows the second sound the pulmonic second sound during inspiration may actually be superimposed on the opening snap (Fig 14)

3 The opening snap of the mitral valve is usually evident in the aortic area and here it follows the second aortic sound at an interval which is not affected by respiration

4 Although a split second sound may occur with or without an accentuated first heart sound the opening snap of the mitral valve occurs only with a good or accentuated first sound

The opening snap can be distinguished from a normal or abnormally accentuated third heart sound on the following bases

1 The third heart sound is loudest near the apex and unless quite loud is not easily heard elsewhere in the precordium Some of the protodiastolic triple rhythms and right sided gallop rhythms may be heard best along the left border of the sternum but these occur infrequently in mitral stenosis

2 The third heart sound normal or abnormal is more widely separated from the second heart sound than the opening snap

3 The third heart sound is of much lower pitch and does not have the sharp character of the opening snap in the fourth left intercostal space

4 An opening snap is associated with a normal and usually accentuated first sound and other evidences of mitral stenosis

An opening snap of the tricuspid valve may occur It is of maximum intensity at the lower end of the sternum and may be well heard or even maximum along the lower right border of the sternum Since the opening snap of the mitral valve is much more common and is widely heard caution must be exercised in making the diagnosis of an opening snap of the tricuspid valve It may be considered when in the presence of other evidence of tricuspid stenosis the opening snap is of maximum intensity at the lower end of the sternum or to the right of the sternum

It should be possible to distinguish the tricuspid opening snap phonocardiographically *since with respiration the snap should* maintain a constant time interval with the pulmonic component of the second sound rather than with the aortic component A phono-

cardiographic study of several patients with tricuspid stenosis associated with mitral stenosis indicates that the opening snap of the tricuspid valve usually occurs just before that of the mitral valve and is difficult to recognize by ear in the presence of an opening snap of

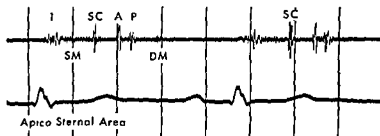


FIG 15 —Systolic click This patient had an atrial septal defect and the midsystolic click (*SC*) was heard only on one occasion The patient had been seen several other times and no systolic click was heard The click was most evident in expiration It was not heard in the pulmonic or aortic region but was confined to the area between the apex and the left border of the sternum

Note the split second sound which is characteristic of atrial septal defect In this condition the split second sound is often evident at the apex in spite of the absence of an accentuated pulmonic sound A middiastolic (*DM*) murmur is frequently heard in the apico sternal region in patients with atrial septal defects

the mitral valve With marked tricuspid stenosis the normal inspiratory splitting of the second sound is decreased possibly because filling of the right ventricle is limited by the stenosis

SYSTOLIC CLICKS

Sharp clicking sounds are sometimes heard during systole They commonly seem to be midsystolic (Fig 15) but may occur in any part of systole Although there is usually a single click two or three closely spaced clicks may occur They show a marked variability in intensity they may be loud one moment and gone the next Such variability may occur with change in position with respiration or with no obvious cause The clicks are usually best heard between the lower end of the sternum and the apex Sometimes very short systolic murmurs start or end with the click

Systolic clicks are commonly heard in apparently normal persons and seem to have no special significance even when heard in diseased hearts They may be pleuropericardial in origin and in

some instances seem closer to the ear than the heart sounds They have been heard in connection with pericarditis and thickened roughened areas on the pericardium have been found in patients who have displayed these clicks

Systolic clicks may be confused with a split second sound or opening snap of the mitral valve Clicks are recognized by their sharp quality timing variability and location The term systolic gallop rhythm commonly used when the clicks are present is a poor one since there is usually no gallop cadence

AUSCULTATORY EVIDENCES OF AURICULAR CONTRACTION

SOUNDS AND MURMURS

1 Phonocardiograms often show some vibrations at the beginning of the first heart sound (Fig 5) These have been attributed to auricular contraction since they occur before the onset of the QRS complex of the electrocardiogram Occasionally in normal persons an auricular sound (also called fourth heart sound) may be audible immediately before the first heart sound (Fig 16) The sound is low pitched and can be heard only if the room is quiet and the bell chest piece is held very lightly on the chest wall (Fig 2 *D*)

The sound produced by the auricles may be increased as a result of auricular hypertrophy increased venous return and changes in ventricular distensibility The auricular sound thus produced is occasionally louder than the first heart sound An auricular sound occurs in many conditions and as one becomes aware of this sound it is heard with increasing frequency

The auricular sound may be produced by either the right or the left auricle A left auricular sound is best heard near the apex and usually in expiration The sound is heard in many patients with hypertension and with coronary artery disease (Fig 16) A right auricular sound is best heard along the left border of the sternum and increases in loudness with inspiration This sound may occasionally be heard in a normal person (Fig 16) It occurs in atrial septal defects (Fig 2 *D*) severe pulmonary stenosis with intact septum pulmonary hypertension and Ebstein s disease

The presence of an auricular sound produces a *presystolic triple rhythm* (p 58) The sound may be mistaken for a component of

a split first heart sound or for a presystolic murmur except that it is short and not in crescendo

2 With delayed atrioventricular conduction, an auricular sound may occasionally be heard before the first sound in the absence of any other evident cardiac pathology (Fig 18 D) The diagnosis of prolonged atrioventricular conduction can sometimes be made on this basis If the factors favoring production of an auricular sound

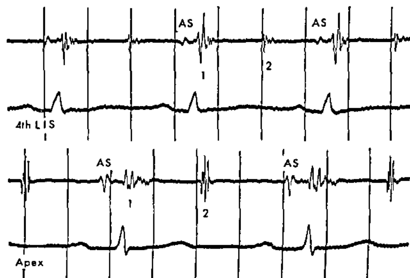


FIG 16—Auricular sounds Upper tracing An auricular sound (*AS*) was heard along the left border of the sternum in a 28 year old woman with a normal cardiovascular system Because of the location in which it was maximum and the increase in intensity with inspiration it was felt *that the sound had its origin on the right side of the heart* Auricular sounds produced in the right side of the heart are commonly heard in patients with atrial septal defects (Fig 2 *D*) and with pure pulmonary stenosis Lower tracing An auricular sound (*AS*) in a 48 year-old man with systemic hypertension and some left ventricular hypertrophy

are present the sound is more easily recognized in the presence of prolonged atrioventricular conduction When conduction is prolonged the auricular sound may at more rapid heart rates be early or middiastolic rather than presystolic

3 In complete heart block a sound produced by auricular contraction can very often be heard It is most evident in early diastole when the auricular contraction is superimposed upon the phase of rapid ventricular filling (Fig 7)

4 In the presence of mitral stenosis auricular contraction pro

duces a presystolic murmur or a presystolic accentuation of a mid diastolic murmur (p 87)

Effect on the First Heart Sound

The intensity of the first heart sound is affected by the time interval between the ventricular contraction and the preceding auricular contraction Changes in the loudness of the first heart sound due to changes in this time relationship can be recognized in complete heart block (p 33) ventricular tachycardia with independent auricular rhythm (p 35) and acute rheumatic fever with changing atrioventricular conduction (p 33)

TRIPLE RHYTHMS (GALLOP RHYTHMS)

A rhythm consisting of three sounds is called a triple rhythm The three sounds are usually the first and second heart sounds and a third sound of varied origin e g an accentuated third heart sound an auricular sound a systolic click If the three sounds are unevenly spaced and of unequal intensity they may give the impression especially at faster heart rates of a gallop It seems unwise to label all triple rhythms as gallop rhythms since most of them do not have a gallop cadence and gallop rhythm has by usage over the years become associated with a specific type of triple rhythm I should like to retain the term *gallop rhythm* for the triple rhythm produced by an extra sound in diastole and associated with a failing myocardium

In the gallop rhythm associated with a failing *myocardium* three factors are usually present (1) the heart rate is over 90 and usually over 100 (2) a sinus rhythm is present the gallop rarely if ever occurs in auricular fibrillation (3) a weakened myocardium is present Because of the rapid heart rate the gallop sound is usually quite close to middiastole it is difficult and useless to attempt to determine by auscultation whether the extra sound is closer to the second heart sound (protodiastolic) or the first heart sound (presystolic) (Fib 17 and 18 *E*) It can be made to sound like either one according to how the rhythm is set up in the listener s mind When the left ventricle is failing the gallop is best heard within the apex but may be widely heard In chronic right ventricular failure the gallop may be equally loud or louder near the *sternum* In acute right heart failure due to a pulmonary embolus the *gallop is most* definite closer to the sternum A pulsus alternans and bundle branch

block are often found when a gallop rhythm occurs with left ventricular failure The gallop rhythm is most commonly seen in failure associated with hypertension chronic coronary artery disease, and myocardial infarction Diastolic murmurs are not usually present

The extra sound in this gallop rhythm is probably produced by the superimposition of the auricular contraction on the phase of rapid diastolic filling thus the two factors that can produce a diastolic sound in the presence of a failing myocardium are merged (summation gallop) The lack of auricular contraction would account for the absence of this gallop in patients with auricular fibrillation When the heart rate slows below 90 the gallop usually disappears but a faint sound may persist which can now be recognized as occurring either early in diastole or in presystole

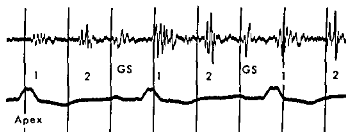


FIG 17 —Gallop rhythm in patient with arteriosclerotic heart disease and failure The rate is rapid The gallop sound (*GS*) occurs in middiastole

Two types of triple rhythm can usually be recognized at slower heart rates a *protodiastolic triple rhythm* (*protodiastolic gallop rapid filling gallop*) in which the extra sound is probably an accentuated third sound and a *presystolic triple rhythm* (*presystolic gallop rhythm auricular gallop*) in which the extra sound is produced by auricular contraction (Fig 18) These rhythms occur at a normal heart rate and do not have the same implication of myocardial failure as the previously described gallop rhythm They are however usually associated with cardiac disease occasionally no other evidence of heart disease is present

A protodiastolic triple rhythm occurs in some patients with mitral insufficiency (Fig 29) hypertension left heart failure and chronic right ventricular failure In right ventricular failure the sound is often well heard near the sternum The protodiastolic sound differs from the normal third heart sound in being louder more widely heard and less influenced by position The abnormal increase in the

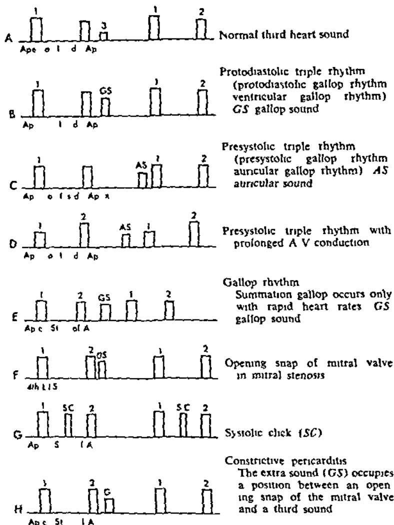


FIG 18 —Table of triple rhythms

third heart sound may be due to one or more of several factors (1) increased flow through the mitral or tricuspid valve resulting in an increase in the forcefulness with which the blood enters the ventricles (2) changes in the muscular tone of the ventricles due to incipient failure of the right or left ventricle and (3) an increased amount of residual blood in the ventricles The intensity of the protodiastolic sound can be decreased by pooling the blood in the extremities by means of tourniquets

In constrictive pericarditis an extra sound may be heard that is closer to the second sound than the usual protodiastolic gallop sound Nevertheless this sound is probably a rapid filling sound rather than an opening snap and may occur early because the resistence of the ventricle to filling is increased by the constrictive pericarditis

Presystolic triple rhythms have been discussed under auricular sounds (pp 55 and 56)

CHAPTER 6

Murmurs General Considerations

PRODUCTION

MOST MURMURS ARE produced by turbulent blood flow The occurrence and degree of turbulence will depend on the velocity of blood the nature and configuration of any obstruction and the viscosity of the blood A brief review of some of these factors is warranted

1 VELOCITY OF BLOOD FLOW —The center part of the blood stream flows faster than the periphery and above a certain velocity eddies form in the current because of this difference in speed of flow Turbulence with resulting murmurs may be produced in a normal heart if the velocity of flow exceeds a critical level Increased velocity of flow may be a partial cause of murmurs heard in exercise thyrotoxicosis and anemia With roughening of a vessel wall the critical speed necessary to produce turbulence is decreased on the other hand a murmur due to obstruction may disappear if the velocity of blood flow becomes less than the critical speed necessary to produce turbulence

2 OBSTRUCTION —A gradual constriction of a vessel will not produce turbulence but a sudden constriction followed by a return to the original size will produce turbulence and a murmur A sudden dilation of a vessel without a previous constriction will produce turbulence in comparison with the dilation the normal vessel is now relatively constricted Mitral stenosis and aortic stenosis constitute a constriction that produces a murmur Similar murmurs may be heard when the left ventricle is dilated and the mitral valve is of normal size or when the aorta is dilated and the aortic valve is of normal

size Here the normal mitral and aortic valves are relatively constricted in relation to the dilated left ventricle and aorta

3 VISCOSITY —The dampening effect of the viscosity of the blood on turbulence possibly accounts for the fact that very few murmurs are normally heard With marked increase in the viscosity of the blood such as occurs in some congenital cardiac anomalies the tendency for production of murmurs is decreased On the other hand the diminished viscosity of the blood in anemia may account in part for the murmurs heard in this condition

4 OTHER SUGGESTED MECHANISMS OF MURMUR PRODUCTION When a regurgitant stream strikes the edge of a valve and causes it to vibrate a murmur is produced in the same manner that a note is produced in a reed instrument The pitch in this type of murmur may be higher than that resulting from a constriction

Collision murmurs may originate when the cardiac or vascular *wall is struck by a current of blood for example when the regurgi*tant current of blood in mitral insufficiency strikes the wall of the left auricle or when a stream of blood through a stenosed aortic or pulmonary valve strikes the vessel wall

Normal structures such as the chordae tendineae or unusual structures such as the so-called ventricular moderator bands may be set into vibration and produce rather musical murmurs It is conceivable that the entire heart or a major section of the heart or great vessels may be set into vibration at its natural frequency by the motion of the blood through it Some such mechanism may play a part in the production of innocent murmurs

5 COMBINATION OF FACTORS —The velocity of blood flow is a factor influencing the occurrence of murmurs with any of the other factors The murmur of mitral stenosis may not be heard at rest but becomes evident with exercise tachycardia and thyrotoxicosis On the other hand a quite evident murmur of mitral stenosis may disappear with decrease in blood flow resulting from decompensation or severe myxedema Most murmurs are louder after exercise In anemia there is an increased velocity of blood flow and a decrease in viscosity both of which would tend to produce murmurs Vibration of the valve together with collision of the regurgitant stream with the heart wall may play a part in producing the murmur of mitral insufficiency Constriction together with the collision of the stream with the vessel wall may play a part in producing the murmurs of aortic and pulmonary stenosis

INSUFFICIENCY AND STENOSIS

INSUFFICIENCY —A valve that cannot close and thus permits the reflux of blood is said to be insufficient Insufficiency or regurgitation may occur either because of pathologic changes involving the valve or because of changes in the supporting structures around the valves The pathologic changes in the valves may vary from minor deformities to marked thickening and shortening of the cusps and chordae tendineae with immobilization of the valves Changes in the supporting tissues consist of dilation of the valve ring and in the case of the atrioventricular valves dilation of the ventricles to the point where the chordae tendineae may not be long enough to permit the valve edges to approximate Involvement of the valve cusps is called *organic insufficiency* When the valve cusps are not involved the terms *functional insufficiency valvular incompetence* and *relative insufficiency* have been used Since the insufficiency is due to pathology involving the heart muscle if not the valves the term *functional* is misleading If *valvular incompetence* were used to refer only to the insufficiency occurring without valvular damage it would be an excellent term for this condition In general however *insufficiency* and *incompetency* are used interchangeably when the pathology is in the valves The term *relative insufficiency* does not give an accurate description of the condition since the insufficiency is not relative to anything *Relative insufficiency* however does not have the objection of implying a functional heart condition and clearly separates the condition from organic mitral insufficiency

STENOSIS —A decrease in the size of a valve due to pathologic changes in the valve produces an *organic stenosis* The term *relative* stenosis is used when the valve itself is normal but when the chamber or vessel beyond the valve is enlarged in this instance the term *relative* is highly appropriate

DESCRIPTION OF MURMURS

TIMING AND DURATION —Systolic murmurs that last throughout systole are called *holosystolic murmurs* If the rate is slow enough variations in the timing of a murmur during systole may be evident The quality of the murmur and the point of maximum intensity however are much more important than timing in the case of systolic murmurs Diastolic murmurs may very definitely occupy one or another portion of diastole and therefore may be described as

early diastolic when they start with the second sound *middiastolic* when there is a short pause after the second sound and *late diastolic* or *presystolic* when due to auricular contraction A *continuous murmur* is heard in systole and diastole and has the same quality in both phases

INTENSITY —Freeman and Levine s* system of grading intensity on a basis of one to six intensity works very well A system based on one to four does not give an adequate range In the one to six system the numbers have the following connotations

One—Very faint
Two—Faint
Three—Moderately loud
Four—Loud
Five—Very loud
Six—Loudest possible

Grade One murmurs can be heard only with concentration in quiet rooms Grade Six murmurs can usually be heard without holding the stethoscope on the chest wall The intensity number should be followed by a six to indicate that the scale of six has been used thus a loud murmur would be indicated by 4/6

The *point of maximum intensity* of a murmur is usually the most important characteristic of the murmur and must be determined accurately Murmurs produced in different portions of the heart are transmitted to different locations on the chest wall

The loudness of a murmur like the loudness of the heart sounds will vary with extracardiac factors This variation must be taken into consideration when correlating the intensity of a murmur with the amount of valvular involvement

It has been noted that the intensity of a murmur will vary with velocity of blood flow Since heart rate is often closely correlated with cardiac output and therefore velocity of flow murmurs may be less evident at slow rates than at rapid rates This fact should be recognized in making comparison of murmurs on different occasions

When the intensity of a murmur during any one cycle increases the murmur may be said to be *crescendo* When it decreases during the cycle it is said to be *decrescendo*

PITCH AND QUALITY —Low pitched murmurs are usually described as rumbling high pitched murmurs as blowing Both low

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Freeman A R and Levine S A The Clinical Significance of the Systolic Murmur Ann Int Med 6 1371 1933

and high pitched murmurs as they increase in loudness become more harsh as a result of the addition of medium and high pitched vibrations Harsh murmurs give an impression of marked turbu lence A medium pitched murmur often gives the impression of roughness Murmurs like heart sounds are essentially noises but when the pitch is clear the sound may have a tonelike or musical quality Murmurs with unusual qualities have been described as sea gull murmurs cooing" murmurs etc

EFFECT OF EXERCISE ON MURMURS —Exercise increases the velocity of blood flow and in most instances the loudness of mur murs Occasionally at rapid heart rates and with the extraneous noises incident to the exercise murmurs may become less evident Exercise is valuable for increasing the intensity of faint murmurs and often important murmurs may be heard only after exercise On the other hand faint murmurs of no significance may be heard in normal persons after exercise They have the characteristics of in nocent murmurs and not of the murmur of mitral insufficiency Exercise is of no value in distinguishing innocent murmurs from organic murmurs since both are usually increased in loudness by exercise For evaluation of the degree of valvular involvement and for comparison of auscultatory findings on different days observa tions made with the patient at rest are more valuable than those after exercise

TRANSMISSION OF MURMURS

Three phases of the transmission of murmurs will be considered (1) factors influencing the location of the point of maximum inten sity of a murmur (2) factors influencing the transmission from the point of maximum intensity (3) changes in the quality of murmurs on transmission

1 FACTORS INFLUENCING THE LOCATION OF THE POINT OF MAXIMUM INTENSITY OF A MURMUR —This point is primarily of course determined by the location of the valve involved The di rection of the stream of blood through the valve however is of great importance because in addition to the vibrations produced in the valve the turbulence produced by the stream accounts for much of the sound Any surgeon can prove this by noting on which side of a pathologic valve the *thrill* is *felt* The importance of the direction of the stream of blood is illustrated in the case of aortic valve lesions In aortic stenosis some of the sound is produced by vibrations set

up in the valves as the blood rushes at high speed past a narrowed orifice Much of the sound is produced in the aorta by turbulence and collision The murmur therefore is better heard in the second and first right interspaces and up into the neck In aortic insufficiency the murmur is produced by the regurgitant stream of blood flowing into the left ventricle Most of the murmur is probably produced in the region of the valve and the murmur is best heard along the left border of the sternum

Another factor influencing the point of maximum intensity is the nature of the tissues between the point of origin of the murmur and the chest wall In an open chest it can easily be shown that the murmur of mitral insufficiency is best heard over the left auricle The left auricle however is separated from the chest wall by sound insulating tissue—the lung The murmur is thus best heard over the left ventricle at the apex In unusual instances in which the left auricle is markedly enlarged and extends anteriorly the murmur may be best heard above the apex where the auricle is close to the chest wall

2 Factors influencing the transmission of a murmur from the point of maximum intensity —The most important factor influencing the transmission of a murmur is the intensity of the murmur The louder a murmur the greater the area over which it will be heard and very loud murmurs will be heard all over the chest and may be transmitted by the bones of the arms to the olecranon process This does not mean that the bones play an important role in the transmission of most murmurs Bones transmit sounds much better than soft tissues because of their rigidity The murmurs however are produced in the soft tissues and a marked difference in the density and structure occurs at the interphase between the soft tissues and the bone Most of the sound when it reaches this interphase will be reflected and very little sound will enter the bone If a very loud murmur is present it may enter the bone in some strength and be widely transmitted by the bone

The direction of the stream which produces turbulence can play a part in the direction in which a murmur is heard The loud systolic murmur heard in aortic stenosis is produced for the main part at the valve and the first part of the aorta Since it is close to the neck and loud it should be and is well transmitted into the neck It would seem quite likely however that the turbulent flow could extend high into the aorta and possibly the larger vessels Thus the sound heard

in the neck may represent not only sound transmitted from the valve and ascending aorta but some sound actually produced in the neck A moderately loud murmur of aortic stenosis is transmitted into the neck better than a louder murmur of patent ductus arteriosus in which the turbulent jet is into the pulmonary artery

3 CHANGES IN QUALITY OF MURMURS WITH TRANSMISSION — The reason why the quality of a murmur may change on transmission has been discussed on pages 12 and 13 Harsh murmurs may lose their harshness and most murmurs become lower in pitch

An additional factor influencing the quality of transmitted murmurs might be considered Most structures when set into vibration have a natural vibration frequency In transmitting sound these structures exaggerate frequencies similar to their natural frequency Frequencies lower than the natural frequency are much better transmitted than higher frequencies The lungs have a natural frequency somewhere between 130 and 180 per minute and they should therefore favor transmission of frequencies at and below this range What the natural vibration frequency of the heart is and how it affects transmission is not known

RELATION OF MURMURS AND THRILLS

A thrill is a palpable manifestation of a murmur the fingers instead of the ears are used to determine the presence of vibration Since the fingers are much less sensitive and discriminating than the ears the vibrations have to be more intense before they are felt The fingers furthermore do not make the fine distinction of pitch and quality A thrill does not tell more about the nature of the underlying lesions than the ear has already learned The association of a thrill with certain lesions such as aortic stenosis in the minds of most people has done more harm than good Many lesions which when severe produce a thrill should and can be recognized by trained auscultation long before the onset of the thrill

Thrills are most commonly felt in association with loud harsh murmurs High pitched murmurs such as the murmurs of aortic and mitral insufficiency are not usually associated with thrills Low pitched murmurs might theoretically be felt better than they can be heard since the fingers are sensitive to vibrations that the ear cannot hear (below 20 per second) There is however little practical value in this theory

CHAPTER 7

Systolic Murmurs

THE QUESTION IS often asked How significant is a systolic murmur? The answer is What systolic murmur and at what stage in its development? The importance of systolic murmurs is often judged by their loudness and it is true that there may be a correlation between the loudness of a systolic murmur and the presence of underlying pathology Some innocent murmurs however may be loud especially in children and some faint organic murmurs may be very significant Nearly all loud organic murmurs other than congenital murmurs must have been faint at one time Other criteria than loudness are better when one is judging the importance of a systolic murmur Most systolic murmurs can be correlated with the underlying pathology and the question then becomes not what is the significance of the murmur but what is the significance of the pathology

THE SYSTOLIC MURMUR OF MITRAL INSUFFICIENCY

TIMING AND DURATION —The murmur of mitral insufficiency is systolic because it is produced by regurgitation through a valve which should be closed during ventricular systole (Fig 20) The murmur usually starts with the first sound and if loud may mask the first sound Louder murmurs last throughout systole (holosystolic) and changes in loudness during systole are not very evident

POINT OF MAXIMUM INTENSITY AND AREA OF TRANSMISSION — Maximum intensity is usually at the apex If the murmur is faint it is heard only in a small area at the apex (Fig 19) When of moderate intensity the murmur may be heard medially in diminishing intensity If the heart is not enlarged the murmur is poorly transmitted

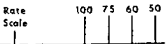
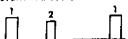



FIG 19—Areas in which murmurs produced at aortic mitral and tricuspid valves are heard when they are faint These are also usually the areas in which these murmurs are of maximum intensity when they are loud With increase in heart size the point of maximum intensity of the mitral and tricuspid murmurs may move laterally

toward the axilla since the lungs effectively dampen it If the murmur is loud and especially if the heart is enlarged so that it is closer to the chest wall laterally the murmur may be clearly heard in the axilla and even posteriorly in the lung bases Even when the murmur is very loud it is poorly transmitted above the third intercostal space into the basal area

A markedly enlarged left auricle that extends anteriorly to the chest wall may cause the maximum intensity to be above and medial to the apex Lung pathology that changes the position of the heart may also change the position of the murmur

PITCH AND QUALITY —When the murmur is of faint or moderate intensity it is characteristically high pitched and has a blowing quality A murmur with this characteristic pitch and quality and of maximum intensity at the apex can be considered as due to mitral insufficiency in the absence of other corroborative findings As the murmur increases in intensity it may remain essentially high pitched or become more harsh because of the addition of medium pitched components This change in pitch and quality in part may be due to the greater amount of sound produced and in part may be associated with differences in pathology of the valve Since the murmur is high pitched the *diaphragm chest piece* is best for its detection

POSITION OF THE PATIENT —The murmur is usually evident in all positions but when faint it may be best heard in the left lateral position immediately after exercise In some patients it is best heard when they are sitting up leaning forward and to the left For faint murmurs the room must be quiet and it may take a minute or two of concentration on systole to hear the murmur One is often asked Why worry about such a faint murmur? Does it have any significance? The answer is definitely Yes The murmur is characteristic and even if it is faint it indicates mitral insufficiency In a child with questionable symptoms of acute rheumatic fever this is a highly important finding

EFFECT OF RESPIRATION —With inspiration the murmur remains unchanged or diminishes somewhat in loudness This is probably due to the increased size of the lungs and their increased dampening effect on the heart sounds and murmurs

CHANGES IN THE HEART SOUNDS —With a fibrotic immobile valve the first sound may be diminished If the valve remains flexible the first heart sound may be normal and actually if some mitral

stenosis is present it may be increased Loud systolic murmurs may mask the first sound so that even if it is normal it may appear to be faint or absent If the bell chest piece is held first lightly and then heavily in contact with the chest wall the change that occurs in loudness of the first sound will make the sound more evident With loud murmurs the second sound may also be masked at the apex

Splitting of the second sound is usually normal but may be increased in severe mitral insufficiency because of rapid emptying of the left ventricle with early closure of the aortic valve

An abnormally loud third heart sound is often present with

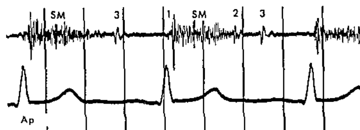


FIG 20—Murmur of mitral insufficiency The holosystolic character of the murmur is illustrated The intensity shows some variation but remains very much the same throughout systole The murmur (*SM*) starts with the first heart sound and continues up to the second heart sound Both heart sounds may be masked by the murmur A third heart sound which is often heard in severe mitral insufficiency is present When the third heart sound is loud it may be mistaken for a masked second heart sound Although the murmur is so loud that the first heart sound is not very evident to the ear the phonocardiogram shows a good first heart sound

marked degrees of mitral insufficiency (Fig 20) This third sound may be louder than and be mistaken for the second heart sound The presence of a third heart sound in a patient with mitral valve involvement is of diagnostic value since it indicates that the valvular lesion is predominantly an insufficiency and not a stenosis (p 91)

RELATION OF LOUDNESS OF MURMUR TO DEGREE OF MITRAL INSUFFICIENCY —It is frequently stated that is some patients no mitral insufficiency is present even though there is a loud murmur and that a marked mitral insufficiency can occur with practically no murmur These situations in my opinion are rare a surprisingly good correlation between the intensity of the murmur and the degree of mitral insufficiency (as determined in operation for mitral steno

sis) will be found if the following factors are taken into considera tion

1 If all the auscultatory phenomena are decreased because of emphysema large breasts or obesity a faint murmur is more sig nificant and must be interpreted in relation to the loudness of the other sounds

2 The murmurs of mitral insufficiency and tricuspid insufficiency must be differentiated (p 74)

3 A loud first sound will mask a following systolic murmur The masking may be overcome in part by using the diaphragm chest piece or by using the bell chest piece with pressure Attention must be directed to systole

4 In the presence of loud or moderately loud systolic murmurs in the aortic area systolic murmurs at the apex must be interpreted cautiously (pp 73 and 80)

If these factors are taken into consideration the correlation be tween the intensity of the murmur and the degree of insufficiency is good although some discrepancies will be observed Some investi gators have found that of patients operated on for mitral stenosis a moderate percentage will have systolic murmurs with no mitral in sufficiency This has been infrequent in my experience Scarring and calcification of the anterior leaflet of the mitral valve have been con sidered a cause of the systolic murmur in such patients and it is possible that this condition may produce a systolic murmur but I doubt that this lesion would produce the characteristic murmur of mitral insufficiency

CONDITIONS PRODUCING MITRAL INSUFFICIENCY —Mitral insuf ficiency resulting from rheumatic or rarely arteriosclerotic involve ment of the mitral valve is referred to as *organic mitral insufficiency* Mitral insufficiency due to conditions which involve the heart but do not affect the valves is referred to as *relative* or *functional insuf ficiency* Relative insufficiency may result from

1 Dilation of the mitral ring or failure of the muscles around the mitral ring to contract properly because of weakness of the muscles or calcification of the annulus fibrosus

2 Dilation of the left ventricle so that the chordae tendineae are too short to permit the valve to close

3 Improper closure of the mitral valve because of the absence of auricular contraction as in auricular fibrillation

DIFFERENTIATION —The systolic murmur of mitral insufficien

cy must be differentiated from other systolic murmurs of maximum intensity at the apex Not many murmurs fortunately other than mitral insufficiency are maximum at the apex and very few of these are high pitched Murmurs having their origin in the aortic valve and possibly the first part of the aorta are usually well heard at the apex and in older patients with deep chests or emphysema the murmur is often louder at the apex than in the second right intercostal space (p 76) Analysis of an apical systolic murmur depends therefore on the findings in the aortic area If no systolic murmur is heard in the second right intercostal space a high pitched systolic murmur of maximum intensity at the apex is almost surely due to mitral insufficiency If a systolic murmur is heard in the second right intercostal space an apical systolic murmur must be interpreted more cautiously This is discussed on page 80

Apical systolic murmurs that are medium pitched and rough rather than high pitched present a problem (1) Many of these murmurs have their origin in the aortic region This is true even when on first examination there appeared to be no murmur in the second right intercostal space A careful re-examination of the aortic region with the patient sitting and holding his breath in expiration may reveal a faint systolic murmur that has the same pitch as the apical murmur (2) Careful check will sometime show that the murmur is actually louder medial to the apex or along the left border of the sternum Such a murmur should be studied for the characteristics of an innocent systolic murmur (p 82) (3) Some medium pitched apical systolic murmurs may be due to mitral insufficiency but other evidence should be sought for confirmation Loud murmurs of mitral insufficiency may be somewhat harsh (4) Some of these medium pitched murmurs are best labelled as of unknown origin

A *late systolic murmur* with a crescendo character is occasionally heard It is often mistaken for a presystolic murmur This murmur is of maximum intensity at the apex and is high pitched Because it is often found in patients with no other evidence of heart disease it has been considered by some to be an innocent systolic murmur (p 82) I have seen this murmur in several patients who showed other evidence of cardiac involvement including left auricular enlargement and feel that it is at times due to some type of mitral valve involvement

THE SYSTOLIC MURMUR OF TRICUSPID INSUFFICIENCY

The murmur of tricuspid insufficiency begins with the first sound and when faint is rather short and decrescendo Louder murmurs last throughout systole The *point of maximum intensity* is to the left of the lower end of the sternum (Figs 19 and 42 *B*) When the murmur is faint it is heard only in this area Louder murmurs, associated with right ventricle enlargement may be heard as far laterally as the anterior axillary line (Fig 44) The point of maximum intensity however remains between the left border of the sternum and the midclavicular line Loud murmurs are transmitted to the right of the sternum but fade rapidly toward the base of the heart

The *pitch* and *quality* of the murmur are similar to those of the murmur of mitral insufficiency—high pitched and blowing The murmur is therefore best heard with the *diaphragm chest piece*

Respiration has a characteristic effect on the murmur of tricuspid insufficiency The murmur increases in loudness often quite markedly with deep inspiration and usually with normal inspiration In some patients the murmur will be heard only during inspiration Augmented filling of the right ventricle during inspiration probably produces this phenomenon With marked tricuspid insufficiency and a loud murmur the effect of respiration on the intensity of the murmur may not be evident This is true especially in the presence of right ventricular failure and increased systemic venous pressure Inspiration may not be able to augment the filling of the right ventricle under these conditions Inspiratory splitting of the second sound may be absent for the same reason

The *first heart sound* in the tricuspid area may be increased normal or somewhat decreased depending on the pathology of the valve and on masking

Differentiation of the systolic murmurs of mitral and tricuspid insufficiency can usually be made on the basis of the effect of respiration and the difference in the points of maximum intensity Mitral murmurs are unchanged or diminished on inspiration and the point of maximum intensity is at the apex or in large hearts lateral to the apex Tricuspid murmurs increase in loudness on inspiration and their point of maximum intensity is closer to the sternum The sign classically associated with tricuspid insufficiency systolic expansion of the neck veins and liver is seen only with severe tricuspid insufficiency is usually associated with right heart failure and need not be present for the diagnosis to be made

Relative tricuspid insufficiency as a result of right heart failure is common In patients with right heart failure it is impossible to determine whether the tricuspid insufficiency is due to organic in volvement of the valve relative tricuspid insufficiency or both In the absence of evidence of increased pressure in the right ventricle tricuspid insufficiency can be recognized on the basis of the char acteristic murmur alone and can be assumed to be of organic origin (Fig 42)

In acute pulmonary infarction the temporary occurrence of the murmur of tricuspid insufficiency (usually not very loud) has not been given the attention it deserves

THE SYSTOLIC MURMUR OF AORTIC STENOSIS AND AORTIC VALVULAR DEFORMITY

Aortic stenosis must involve more than 75% of the opening be fore changes in the pulse wave and blood pressure occur Minor grades of deformity may produce a systolic murmur with little or no change in the circulatory dynamics Many murmurs may represent roughening or perhaps mild deformity of the valve without any sig nificant stenosis one hesitates therefore to call this murmur the murmur of aortic stenosis It is in these cases that the term aortic valvular deformity is used

TIMING AND DURATION —The murmur of moderately severe and severe aortic stenosis builds up to a peak in midsystole and then de creases it is faint or absent by the time the aortic second sound occurs The murmur recorded on phonocardiograms thus has a diamond shape (Fig 21) The ear can learn to recognize this very characteristic sound With lesser degrees of aortic deformity the murmur reaches a peak earlier in systole

POINT OF MAXIMUM INTENSITY AND AREA OF TRANSMISSION — The point of maximum intensity is usually in the second or first right intercostal space (Figs 19 and 22) A murmur well heard in the first right intercostal space is likely to be of aortic origin Murmurs of only moderate intensity are usually well transmitted to the apex and the neck When the murmur is very loud it is heard over the entire thorax and as far down as the olecranon process of the ulna In older patients with deep chests and in the presence of emphyse ma the murmur is often louder at the apex than in the second right interspace In transmission from the base to the apex there is often an area along the left border of the sternum where the mur

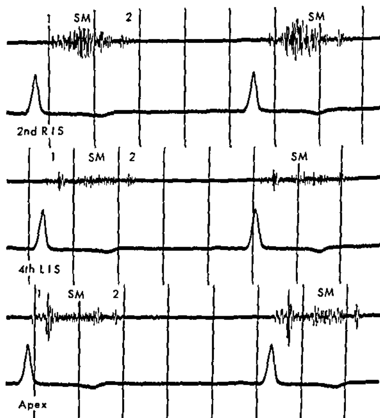


FIG 21 —Murmur of aortic stenosis This murmur (*SM*) is loudest in the second right intercostal space (upper tracing) and in this area it often shows its characteristic "diamond shape" on the phonocardiogram Note that the murmur stops well before the second heart sound and that if a second heart sound is present it can usually be heard although if the murmur is very loud there may still be some masking The murmur is heard well at the apex (lower tracing) and in this area has a quality similar to that in the second right intercostal space In the fourth left intercostal space (middle tracing) the murmur is not as loud as it is at the apex Faint murmurs may be heard at the second right intercostal space and at the apex and not be evident along the left border of the sternum

mur is less intense than it is at the apex or base (Fig 22) Because of changes during transmission (p 67) the murmur may sound somewhat less harsh at the apex than at the base The question of two separate murmurs may thus be posed and at times it may be difficult to decide this point Usually however the rather characteristic quality and pitch of the aortic murmurs will still be recognizable at the apex

PITCH AND QUALITY —Faint murmurs are rough and of medium pitch louder murmurs are harsh and often associated with a thrill Either chest piece may be satisfactorily used At times the murmur is so loud that it can be heard with the stethoscope away from the chest wall

POSITION OF THE PATIENT —The murmur is heard best with the patient sitting up and leaning forward and with the breath held in expiration

CHANGES IN HEART SOUNDS —With advanced degrees of valvular stenosis the second heart sound may be entirely absent in the second right intercostal space with lesser degrees it may be normal or diminished Even when no second sound is heard at the base a second sound of aortic origin may at times be heard at the apex indicating a masking of the sound at the base Sometimes because of some flexibility of the valve a relatively good second sound may be heard in marked stenosis especially congenital aortic stenosis An accentuated pulmonic sound may occasionally account for a second sound heard in the second right intercostal space When both aortic and pulmonic second sounds are heard either normal splitting or paradoxical splitting (Figs 11 and 12) may occur

A third heart sound is at times heard at the apex

RELATION OF INTENSITY OF MURMUR TO SEVERITY OF LESION — Several factors disturb this relationship

1 In persons with deep chests or with emphysema the intensity of all sounds at the base may be diminished and severe aortic stenosis may exist with a faint murmur which is easily overlooked The murmur in these patients may be better heard at the apex and mistaken for that of mitral insufficiency The quality of the murmur at the apex is a clue to listen more carefully at the base

2 In the presence of marked or moderate aortic insufficiency the amount of blood flowing through the valve during systole is increased and flows by the deformed valve at an increased velocity This velocity of blood flow may increase the loudness of the mur

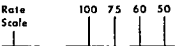


FIG 27—Systolic murmur of aortic stenosis (The figures in parentheses refer to the lines on the chart) This is a harsh systolic murmur which reaches its peak in mid systole and usually fades out so that it disappears before the aortic second sound It is of maximum intensity in the f t

(?) and second (3) right intercostal spaces It is well heard at the apex (9) Along the left border of the sternum (4) the murmur usually is less intense than it is at the apex The murmur is well heard on the right side of the neck (1) The first heart sound is often split (9) A paradoxical splitting of the second heart sound occurs in some patients It occurs usually in patients with severe stenosis in whom an aortic second sound is still present The splitting is more evident on expiration (7) than on inspiration (6) A third heart sound is frequently heard at the apex (9)

In this chart a faint high pitched early diastolic murmur (slight aortic insufficiency) is shown in the third left intercostal space (8) This is frequently heard in patients with aortic stenosis

mur beyond what would be expected from the degree of stenosis

3 With the onset of failure arrhythmia, or shock the murmur may decrease markedly in loudness

OCCURRENCE —Rheumatic fever is the most common cause of aortic valvular deformity and aortic stenosis Calcific aortic stenosis occurs in an older age group and may be of rheumatic origin Congenital aortic stenosis occurs more frequently than has been recognized in the past

DIFFERENTIATION —Differentiation from the more common basal systolic murmur associated with arteriosclerosis and hypertension is given on page 81 When a murmur of mitral insufficiency is present by itself its recognition is no problem Difficulty arises when a classic murmur of aortic stenosis is present and one must decide whether there is also a murmur of mitral insufficiency The murmur of aortic stenosis is well transmitted to the apex whereas the murmur of mitral insufficiency is poorly transmitted to the aortic region The presence of a murmur of similar quality in the two areas therefore usually leaves little doubt that there is an aortic lesion It may be difficult or impossible however to rule out by auscultation the simultaneous presence of a mitral insufficiency When the aortic murmur is not too loud and the murmurs are of different quality both lesions are probably present

THE BASAL SYSTOLIC MURMUR ASSOCIATED WITH ARTERIOSCLEROSIS AND/OR HYPERTENSION

A basal systolic murmur is probably the most common murmur heard in older people and is frequently associated with other evidence of arteriosclerosis or hypertension The aorta is often widened and tortuous The murmur is of maximum intensity in the second right intercostal space (Fig 23) It is of medium pitch and rough rather than harsh like the murmur of aortic stenosis The murmur is not usually very loud and does not get as loud as the murmur of aortic stenosis but an overlap occurs both in intensity and in quality The murmur is best heard when the patient is sitting up and leaning forward Either the diaphragm or the bell chest piece may be used

The murmur is often *well transmitted to the apex* and it may be difficult to decide whether an apical systolic murmur in these patients is transmitted from the aortic area or whether it represents a mitral insufficiency If the aortic systolic murmur is moderately loud and the apical *systolic murmur does not have the high pitch and blowing*

quality of a mitral insufficiency murmur the murmur should usually be considered as having been transmitted from the aortic area Since all the sounds may be faint at the base because of emphysema or a deep chest the murmur may be louder at the apex than at the base Transmission into the neck occurs but is not marked possibly be cause the murmur is usually not very loud

The second sound is of normal or increased loudness

Factors that may play a part in the production of this murmur are

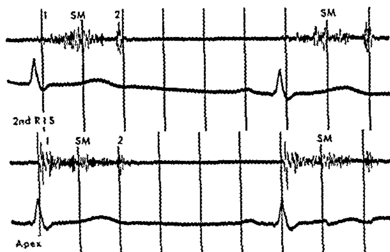


FIG 23 —Basal systolic murmur associated with arteriosclerosis This murmur (*SM*) is best heard in the second and first right intercostal spaces It is often transmitted to the apex It is usually not as long a murmur as that of aortic stenosis and reaches a peak earlier in systole

arteriosclerotic roughening of the aorta and aortic valves and *rel ative aortic stenosis* resulting from dilation of the aorta (p 61)

This murmur will not be confused with that of aortic stenosis when the latter is loud and harsh When the murmurs are of moderate in tensity the following factors help to distinguish the two

1 The age group in general is different although calcific aortic stenosis often occurs in the same group

2 The presence of a dilated aorta hypertension or arterioscle ro sis favors the diagnosis of relative aortic stenosis The presence of other murmurs (indicating aortic insufficiency or mitral stenosis) favors the diagnosis of organic aortic stenosis

3 The second aortic sound is of normal or increased loudness in

patients with arteriosclerosis or hypertension and is often diminished or absent in patients with aortic stenosis

INNOCENT SYSTOLIC MURMURS

Innocent systolic murmurs are systolic murmurs not due to any recognizable heart pathology As here used however, the term innocent systolic murmurs refers to a clear cut group of murmurs with definite characteristics that permit their recognition The term does not include murmurs that are clearly recognized as being produced in some valve or congenital defect but are innocuous (innocent) in that there is no other evidence of cardiac involvement Nor is the term to be used as a catchall for murmurs of which the origin is unknown but which do not have the characteristics of the murmurs to be described

Innocent systolic murmurs are also called *accidental murmurs* and *physiologic murmurs* Unfortunately they are also often called *functional murmurs* As previously noted (p 63) the term functional is commonly used to describe murmurs that occur in diseased hearts but are not due to organic valvular involvement e g functional pulmonary insufficiency in pulmonary hypertension Because the term functional is ambiguous and often misleading it is best not used at all The innocent murmurs here described should not be called functional murmurs When the murmur is produced at a valve because of myocardial rather than valvular involvement the term *relative* is preferred to functional e g relative mitral stenosis or relative mitral insufficiency If the murmur does not fit any of the described murmurs and its cause is unknown it is best labeled a murmur of unknown etiology

The innocent systolic murmurs are the most common systolic murmurs heard in children and their main importance is that they must be separated from those murmurs that indicate organic valvular pathology or congenital heart disease Some studies of children demonstrate that as many as 50% may show at one time or another an innocent murmur These murmurs may persist for years or disappear quickly The exact mode of production of most of them is not known

Innocent systolic murmurs tend to be short and rarely extend through entire systole (Fig 24 *B*)

POINT OF MAXIMUM INTENSITY —Innocent systolic murmurs may be separated into two groups (1) those of maximum intensity

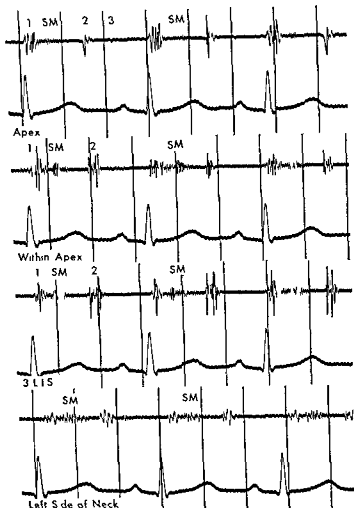


FIG ₂4 *A*—Innocent systolic murmur This murmur was of maximum intensity between the apex and the left border of the sternum although it can be noted that the intensity within the apex (second tracing) and in the third left intercostal space (third tracing) is similar and it would be difficult to say that the murmur was louder in one area than in the other It was definitely not loudest at the apex (first tracing) Note the intensity in the left side of the neck (fourth tracing)

in the apico sternal region (the area from within the apex to the left sternal border) and (2) those of maximum intensity in the second left intercostal space In the apico sternal group, localization of the exact point of maximum intensity may at times be difficult because the murmur may be of almost equal intensity over much of the apico sternal area (Fig 24 *A*) The point of maximum intensity will vary somewhat with the patient s position and is about an intercostal space lower when the patient sits up *These murmurs are not of maximum intensity at the apex*

AREA OF TRANSMISSION —A very interesting and important differentiating characteristic of the apico sternal murmurs is that these

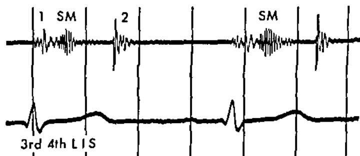


FIG 24 *B*—Innocent systolic murmur Note the uniform vibrations that are characteristic of those innocent systolic murmurs that have a "vibratory" quality Also note that the murmur is short and that it is mainly in the first half of systole

murmurs considering their intensity are heard in a wide area from the point of maximum intensity A murmur of moderate intensity and maximum in the third left intercostal space may be heard at the apex in the aortic area and usually into the neck Many innocent systolic murmurs are especially well transmitted into the neck actually much better than most organic murmurs e g the murmur of patent ductus arteriosus Because they may be heard at the apex they will be recorded as apical systolic murmurs if the precordium is not carefully investigated to determine the point of maximum intensity These murmurs are almost always heard in the pulmonic area no matter where they may be most intense This is not true of the systolic murmur of mitral insufficiency

An innocent murmur that is of maximum intensity in the second left intercostal space tends to be localized and does not show the wide transmission of the apico sternal murmurs

LOUDNESS —Some of the innocent murmurs may be quite loud in children Exercise as it does for most murmurs increases the loud ness of innocent murmurs and is of no value in differentiating them from other murmurs Fever and tachycardia will increase the loud ness of a murmur and often a murmur will be heard only when these conditions are present

PITCH AND QUALITY —Innocent systolic murmurs are of medium pitch and are not usually harsh They are *not* high pitched The term vibratory has been used and is descriptive of the quality of many of the innocent systolic murmurs heard in the apico sternal region (Fig 24 *B*) To realize the true quality of the murmur one must listen to it with a bell chest piece held lightly With heavy pressure on the bell the murmur is usually greatly diminished but often some high pitched components remain which may resemble the murmur of mitral insufficiency The diaphragm chest piece may also be misleading with regard to the quality of the murmur

Most innocent murmurs of maximum intensity in the second left intercostal space and some murmurs of maximum intensity in the apico sternal region may be rough rather than vibratory

POSITION OF THE PATIENT —Innocent systolic murmurs are usu ally best heard when the patient is lying down but may occasionally be heard better when the patient is sitting up The murmur will often increase at the apex in the left lateral position in a manner suggesting the murmur of mitral insufficiency However the murmur will usu ally be found to have also increased in intensity along the left border of the sternum and will be louder in that region than at the apex

EFFECT OF RESPIRATION —Held expiration will increase these murmurs especially those that are loudest in the pulmonic area There are few children in whom a faint systolic murmur will not be heard in the pulmonic area if the child expires forcibly and stops breathing for 15 or 20 seconds

CHANGES IN THE HEART SOUNDS —With innocent systolic mur murs there are no changes in the heart sounds The clear-cut pres ence of heart sound changes especially an abnormally split or ac centuated second sound should therefore make one consider the possibility of a congenital heart murmur

DIFFERENTIATION —The differentiation of these murmurs from the murmurs produced by rheumatic heart disease is usually easy and depends on the *point of maximum intensity quality* and *trans mission* which as has been indicated are quite different from those

of the murmurs described up to this point In doubtful cases the presence of a clear cut diastolic murmur is helpful in deciding whether the murmur is innocent or organic However innocent murmurs may occur in the presence of organic murmurs and can often be recognized as such In some normal children after exercise the third heart sound at the apex may be prolonged and give the impression of a middiastolic murmur If this sound is mistaken for a mid diastolic murmur an innocent systolic murmur may be given more importance than it deserves

The differentiation of innocent systolic murmurs and some systolic murmurs occurring in acyanotic congenital heart disease may present some problems

1 An innocent systolic murmur of maximum intensity in the second left intercostal space may sound very much like the murmur of an atrial septal defect Here the helpful differentiating point is the presence of a split second sound in atrial septal defects (Fig 35)

2 An innocent systolic murmur of maximum intensity in the third or fourth left intercostal space may need to be differentiated from the murmur of a small ventricular septal defect If the innocent murmur has a vibratory quality this is helpful The point of maximum intensity of an innocent murmur is not as sharply localized as that of the murmur of a ventricular septal defect An innocent murmur is more widely heard than a murmur of a ventricular septal defect of comparable intensity Finally innocent systolic murmurs are much more common than ventricular septal defects nevertheless differentiation may at times be impossible

CARDIOPULMONARY MURMUR

The cardiopulmonary murmur is frequently mentioned in the literature but does not seem to occur commonly although it may be that it is so easily recognized that one soon learns to ignore it Along the borders of the heart where the respiratory sounds can be heard along with the heart sounds the inspiratory sound will seem at times to be broken up and heard only during systole It gives the impression of a faint high pitched murmur resembling the murmur of mitral insufficiency This apparent murmur occurs only during inspiration or at least is much louder during inspiration Having the patient hold his breath does away with the murmur and is the most important means of recognizing the murmur if there is any doubt The heart action against the adjacent lung tissue may account for the production of this apparent murmur

CHAPTER 8

Diastolic Murmurs

THE DIASTOLIC MURMUR OF MITRAL STENOSIS

TIMING AND DURATION —The murmur of mitral stenosis is pro duced by the flow of blood into the ventricles during diastole and is most intense when the flow is greatest Figure 25 *A* shows the ventricular volume curve in a normal heart The greatest filling oc curs during the phase of rapid inflow following the opening of the atrioventricular valves By the time of the auricular contraction most of the ventricular filling has occurred and little is added by auricular contraction If the mitral valve becomes mildly stenotic a murmur is heard during the phase of rapid inflow (Fig 25 *B*) If the heart rate is slow this murmur is at first short Since the mur mur occurs an appreciable time after the second sound it has been called a middiastolic murmur A murmur may occur with auricular contraction at this stage especially at more rapid rates since auricu lar contraction now occurs before ventricular filling is complete at slow rates only the middiastolic murmur may be present

As the degree of stenosis increases the filling of the ventricle is slower and the murmur becomes longer Since the ventricle is less likely to be filled by the time of auricular contraction the increased flow produced by auricular contraction produces a murmur (Fig 25 *C*) This murmur is called presystolic and to the ear is crescendo in character ending in the first sound As the stenosis increases the middiastolic and presystolic components blend into one murmur which all too often is called presystolic when it is really middiastolic and presystolic (Fig 26) At rapid rates the two murmurs telescope so that the murmur is shorter and mainly presystolic

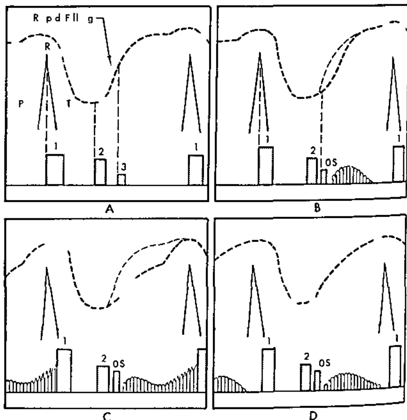


Fig 25 —Mitral stenosis—relation of ventricular filling to heart sounds and murmurs Upper dashed line ventricular volume Middle curve electro cardiogram Lower line heart sounds and murmurs

A normal The first heart sound usually begins near the peak of the R wave of the electrocardiogram The second heart sound indicates the end of systole and usually occurs at the end of the T wave The third heart sound occurs near the end of rapid ventricular filling *B* early mitral ste nosis Because of changes in the mitral valve the first heart sound is some what increased and a sound is produced when the mitral valve opens (*OS*) This opening snap occurs shortly after the second sound Ventricular filling is slightly delayed and a murmur is produced during the period of rapid ventricular filling The fine dashed line indicates normal ventricular filling Because the stenosis is mild ventricular filling although somewhat slowed is adequate and if the heart rate is slow the ventricles are full by the time auricular contraction occurs Auricular contraction adds little to the flow and a presystolic murmur may not be present or may be very faint *C* severe mitral stenosis The first sound is accentuated and is delayed owing to the high pressure in the left auricle and the time necessary for the ven tricle to build up a pressure equal to that in the auricle Because of this delay in the first sound systole is shortened This is a characteristic auscul

In auricular fibrillation (Fig 25 *D*) only the middiastolic component is present If however a loud snapping first sound occurs while the middiastolic murmur is fairly loud the impression the ear gets is one of a crescendo presystolic murmur

It is important to repeat that the earliest murmur of mitral stenosis is nearly always a middiastolic murmur although occasionally only a presystolic murmur can be heard However it is always dangerous to make the diagnosis of mitral stenosis on what appears to be a presystolic murmur alone since other sounds occurring at the time

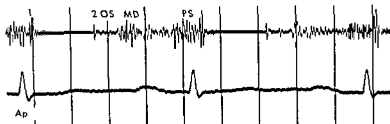


Fig 26 —Diastolic murmur of mitral stenosis The second sound is followed by a faint opening snap (*OS*) Diastole is long and the middiastolic murmur (*MD*) begins to fade before auricular contraction produces the presystolic (*PS*) murmur

of the first sound can give the impression of a presystolic murmur e g an auricular sound or a split first sound

Maximum intensity and area of transmission —The murmur of mitral stenosis is of maximum intensity just within and above the apex (Fig 19) It is generally confined to a rather small area even when the murmur is quite loud in this area It is heard at the base only if very loud and then not commonly With enlargement of the right ventricle the left ventricle and auricle may be pushed laterally and posteriorly and the murmur in these cases will often be heard in the axillary or midaxillary line

Pitch and quality —When faint or moderately loud the middiastolic murmur is low pitched and rumbling As it becomes louder it becomes more harsh Because the murmur is low pitched it is

tatory feature in severe mitral stenosis The opening snap is loud Because of the stenosis a murmur starts shortly after the opening of the mitral valve and persists throughout diastole At the time of auricular contraction the ventricles are still not full and auricular contraction increases the flow producing a harsh presystolic murmur which ends with the accentuated first heart sound *D* with the onset of auricular fibrillation the presystolic murmur produced by auricular contraction disappears

best heard with the bell held very lightly on the skin in fact when the murmur is faint it will be heard only with the bell held lightly The use of the diaphragm chest piece or heavy pressure on the bell will completely obliterate a faint murmur that is clearly heard with the bell held lightly (Fig 2 B) Since even faint middiastolic murmurs are highly significant the skilled use of the bell is one of the important assets in auscultation As the murmur becomes louder it becomes more harsh and may be heard equally well with bell or diaphragm The presystolic murmur is more harsh than the mid diastolic murmur and is heard almost equally well with the bell or diaphragm

POSITION OF THE PATIENT —The murmur is best heard when the patient is in the recumbent position even when the murmur is loud in this position it may not be heard at all in the upright position To listen to the patient only in the upright position therefore is to ignore one of the most important murmurs in cardiac auscultation The murmur is often better heard in the left lateral position but not always Nevertheless an examination for this murmur is incomplete unless the patient is turned into the left lateral position The murmur is usually increased for a few beats just as the patient is turned from recumbent to the left lateral position or from the left lateral to the recumbent and it is therefore important to listen while turning the patient

EFFECT OF RESPIRATION —The murmur is either unchanged by respiration or somewhat diminished on inspiration

CHANGES IN HEART SOUNDS —Mitral stenosis is usually associated with an accentuated first heart sound When the first sound is loud there is usually an opening snap of the mitral valve (p 49) If the valve and chordae tendineae are markedly fibrosed and the cusps are immobile the first sound is diminished in loudness and usually there is no opening snap of the mitral valve The second heart sound may be accentuated in the pulmonic area but this accentuation is due to an increase in pulmonary artery pressure and is a late change The presence or absence of an accentuated pulmonic second sound gives information regarding pulmonary artery pressure and not regarding the presence or absence of mitral stenosis The frequent occurrence of an opening snap of the mitral valve accounts for the common impression that the second heart sound is abnormally split in mitral stenosis The splitting is usually of normal degree

A third heart sound at the apex is rarely heard if there is a significant degree of mitral stenosis The production of a third sound depends in part on rapid ventricular filling This cannot occur with tight mitral stenosis The presence of a third heart sound at the apex therefore accentuates the importance of a systolic murmur and indicates that the insufficiency is the predominant lesion A right sided triple rhythm may occur in mitral stenosis with right ventricular failure however this third sound will not usually be mistaken for that heard in mitral insufficiency Since an opening snap of the mitral valve can occur in the presence of mild mitral stenosis it may occasionally be heard in the presence of mild stenosis with fairly marked mitral insufficiency and a third heart sound

EFFECT OF EXERCISE —Since exercise increases the cardiac output and the speed of blood flow it will increase the intensity of the murmur of mitral stenosis or will bring out a murmur not evident when the patient is at rest Since even a faint murmur brought on by exercise is significant it is important if there is any suspicion of mitral stenosis that the patient be exercised

RELATION OF INTENSITY OF MURMUR TO SEVERITY OF LESION — To make any correlation the following factors must be considered

1 The general intensity of all the sounds and murmurs must be compared since a heavy chest wall or emphysema may dampen a loud murmur In marked emphysema a significant mitral stenosis may be present with a very faint murmur which because of the voluminous lungs is heard much closer to the sternum than normally

2 The cardiac output mainly as judged by heart rate must be considered The increase in heart rate that occurs with excitement exercise thyrotoxicosis and fever is associated with a marked increase in intensity of the murmur A decrease in output such as occurs in decompensation may make the murmur much less evident or may make it disappear The murmur is less evident at rest and in myxedema For comparison of the murmur on different occasions it is best to use the intensity at rest

3 When an enlarged right ventricle pushes the left auricle and left ventricle laterally and posteriorly the murmur is heard faintly in the midaxillary line and may be completely overlooked

4 When a stenosis becomes very marked the stream producing the murmur may be small and the murmur actually less evident

5 The murmur produced by a given degree of mitral stenosis will

be increased by a significant degree of mitral insufficiency A significant degree of mitral insufficiency implies of course that the stenosis *is not marked* *In mitral insufficiency the flow through the mitral* valves is increased since there must be enough flow during each diastole to make up for what is to be regurgitated on the next beat There is also an increase in the size of the left ventricle in mitral insufficiency which would make the stenosis relatively greater In the presence therefore of mitral insufficiency a murmur of mitral stenosis is increased out of proportion to the degree of stenosis

In spite of the many factors influencing the relation between the intensity of the murmur and the degree of mitral stenosis there is usually a rough correlation

The *duration of the murmur* should be given as much attention as the intensity since it is closely correlated with the degree of stenosis and may be subject to fewer modifying factors

DIFFERENTIATION —The murmur of mitral stenosis can be separated from the early diastolic murmurs of aortic and pulmonary insufficiency with no difficulty The main problem is differentiating it from the murmurs of tricuspid stenosis and of relative mitral stenosis These will be discussed below

EFFECT OF MITRAL COMMISSUROTOMY —See page 146

THE DIASTOLIC MURMUR OF TRICUSPID STENOSIS

The diastolic murmur of tricuspid stenosis usually has the same timing and much the same pitch and quality as that of mitral stenosis occasionally however the murmur seems higher pitched and earlier in diastole and may somewhat resemble the early diastolic murmur of aortic and pulmonary insufficiency The murmur is best heard in the recumbent position with the bell chest piece The first sound is less commonly accentuated than in mitral stenosis

Tricuspid stenosis practically always occurs in association with mitral valvular lesions which may overshadow it However the presence of a tricuspid stenosis can usually be recognized even in the presence of a mitral stenosis by the *characteristic location* of the murmur and its *reaction to respiration* The murmur is best heard just to the left of the lower end of the sternum and in the fourth left intercostal space near the sternum It is usually fairly well localized and does not extend far to the left even when loud and when the right side of the heart is quite enlarged If the murmurs of mitral stenosis and tricuspid stenosis are both present two areas of maxi

mum intensity can often be determined one close to the sternum and the other at the apex With cardiac enlargement both areas may be shifted somewhat to the left

Like the murmur of tricuspid insufficiency the murmur of tricuspid stenosis is markedly increased in loudness with inspiration It may be several times as loud in inspiration as in expiration and may be heard only in inspiration The murmur of mitral stenosis will be essentially unchanged or slightly decreased on inspiration

Tricuspid insufficiency will increase the loudness of the murmur of a mild tricuspid stenosis This effect results from the increased

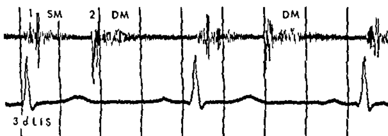


FIG 27 —Diastolic murmur of aortic insufficiency The high pitched diastolic murmur (*DM*) starts with the second sound It is loudest early in diastole and in this case is heard throughout diastole in diminishing intensity A moderately loud medium pitched systolic murmur (*SM*) is present in this patient The second sound is normal or slightly accentuated

flow through the tricuspid valve during diastole and the relative stenosis produced by the dilation of the right ventricle a stretching and distortion of the valve has also been suggested With marked tricuspid insufficiency a diastolic murmur can occur even in the absence of stenosis Since some tricuspid insufficiency is nearly always associated with tricuspid stenosis caution must be exercised in estimating the degree of tricuspid stenosis from the loudness of the diastolic murmur

A palpable shock in the jugular vein synchronous with the presystolic portion of the murmur may if present be a valuable confirmatory sign

THE DIASTOLIC MURMUR OF AORTIC INSUFFICIENCY

TIMING AND DURATION —The maximum intensity of the diastolic murmur of aortic insufficiency occurs immediately after the second sound as the pressure in the aorta falls and the ventricles fill the murmur decreases in intensity (Fig 27) Since it is loudest in early

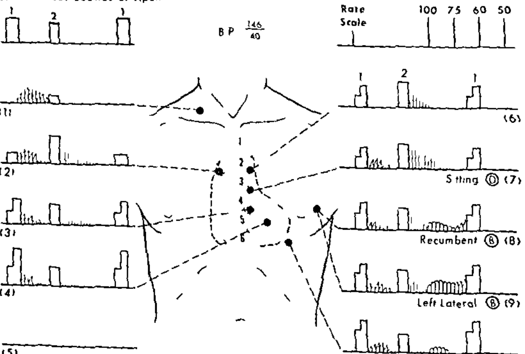


Fig 28—Severe aortic insufficiency A mild mitral stenosis or an Austin Flint murmur is also pre ent The numbers in parenthe es refer to the l nes on the chart The following features re ho n

a) A high pitched diastolic murmur starting with the second sound and diminishing in in-
tensity is the *characteristic finding* The murmur is loudest in the third left intercostal space (7)
and is usually well heard in the fourth left intercostal space (3) *When loud it is heard* to a less-
er degree in other areas of the precordium It is occasionally loudest in the second right intercostal
space

b) When the heart is very large the early diastolic murmur is often well heard in the anterior
axillary line above the apex (8) (9) It is often of greater intensity in this area than it is in any
other area except the third left intercostal space

c) At the apex and laterally from the apex a rumbling middiastolic murmur and a presys-
tolic murmur are present (8) (9) (10) This middiastolic and presystolic murmur may represent
a mild degree of organic mitral stenosis or it may be an Austin Flint murmur If the cardiac
lesion is due to rheumatic fever one must assume that the middiastolic murmur is probably due
to mitral valve involvement

d) A rough systolic murmur is usually present in the second right intercostal space (2) This
may occur even in the absence of aortic stenosis and may be due in part to roughening of the
aortic valves and in part to a *relative* aortic stenosis produced *by some* aortic dilation and by the
marked increase in flow through the aortic valve

e) Although the heart sounds are often unchanged a split first sound with a second com-
ponent louder than the first component is often present

diastole it is considered an early diastolic murmur even though it may last throughout diastole Phonocardiograms occasionally show that the murmur has a short period of crescendo and then becomes decrescendo the ear can occasionally distinguish this sequence but essentially the murmur can be considered decrescendo The murmur may be very short or may last throughout diastole

PITCH AND QUALITY —The murmur is high pitched and blowing and usually remains so even when loud When aortic insufficiency is very severe the murmur may be harsh and surprisingly short for the degree of insufficiency A loud high pitched musical murmur may occur when the insufficiency is associated with a retroverted cusp

Since the murmur is high pitched the diaphragm chest piece is essential when the murmur is faint The room must be quiet and at tention must be directed at early diastole A normal or accentuated second sound may mask a faint murmur unless the latter is carefully sought

POINT OF MAXIMUM INTENSITY —This is usually in the third left intercostal space next to the sternum and when the murmur is faint it may be heard only in this area (Fig 19) When the aorta is dilated the murmur may be loudest in the second right intercostal space When the murmur is loud and has its point of maximum in tensity to the right of the sternum the diagnosis of a dissecting aneurysm or an aneurysm of the sinus of Valsalva should be con sidered Occasionally when all sounds are faint at the base the mur mur may be most intense to the left of the lower end of the sternum or at the apex

AREA OF TRANSMISSION —When the murmur is loud it may be heard over most of the precordium In some cases when the left ventricle is quite enlarged the murmur may be loudest along the left border of the sternum but is also very well heard in an area above the apex in the anterior axillary region (Fig 28) Between these two areas the murmur may not be heard at all or may be only faintly heard With transmission the murmur loses some of its characteristic high pitch but is not difficult to recognize because of its timing

When the aortic insufficiency is marked and associated with a moderately loud systolic murmur of aortic stenosis an unusual aus cultatory phenomenon may occasionally be heard in the left anterior axillary region In this area the transmitted systolic and early dias

tolic murmurs combine with an apical middiastolic murmur due to relative mitral stenosis (p 98) to give the impression of a continuous murmur similar to that heard in arteriovenous fistulae The lowered pitch of the early diastolic murmur resulting from transmission favors this impression

POSITION OF THE PATIENT —Older patients should be sitting up and leaning forward with respiration held on expiration In younger patients the murmur is often as well or better heard in the recumbent position

CHANGES IN HEART SOUND —The aortic second sound is frequently increased in aortic insufficiency (pp 43 44) With syphilitic involvement of the aorta the second sound may also have a resonant or tympanitic quality If there is an associated aortic stenosis the aortic second sound may be diminished

RELATION OF INTENSITY OF MURMUR TO SEVERITY OF LESION — When the insufficiency is mild a faint murmur may be the only evidence of the lesion When a lesion is severe enough to produce peripheral signs of aortic insufficiency the murmur is usually more intense and prolonged On occasion marked peripheral evidence of insufficiency may be associated with a very short harsh murmur

OCCURRENCE —The diastolic murmur of aortic insufficiency is most commonly heard in association with rheumatic heart disease Syphilitic aortic insufficiency is becoming less frequent Faint murmurs are heard sometimes in hypertension and less commonly in arteriosclerotic heart disease The sudden appearance of this murmur in a patient with hypertension should make one think of a dissecting aneurysm Aortic insufficiency occurs with some congenital heart defects especially in association with a bicuspid aortic valve A loud high pitched musical murmur associated with a retroverted cusp or possibly a ruptured cusp occurs primarily in syphilitic aortitis but is also heard occasionally in rheumatic aortic insufficiency or bacterial endocarditis and rarely in arteriosclerosis This loud musical murmur may persist for years or gradually be replaced by the usual murmur of aortic insufficiency

APICAL DIASTOLIC MURMURS NOT ASSOCIATED WITH ORGANIC MITRAL STENOSIS

Murmurs resembling the murmur of mitral stenosis in quality and timing occur in a number of conditions in which an organic mitral stenosis is not present In general one or both of two con

ditions are present when these murmurs are found an increase in the size of the left ventricle or an increased flow of blood through the mitral valve A normal mitral valve may be relatively stenotic if there is a markedly enlarged left ventricle An increased flow through the mitral valve occurs in such conditions as patent ductus arteriosus ventricular septal defect and mitral insufficiency Poor tone of the muscle of the left ventricle may at times play a part e g in rheumatic carditis

A similar murmur may be heard in relative tricuspid stenosis when the same factors are present

The murmur is most often seen in children and this is probably due in part to the fact that blood flow through the mitral valve in children seems more likely to produce sound Phonocardiograms in normal children may show a few vibrations during diastole in association with a third heart sound

The *timing* of the murmur is similar to that of the murmur of mitral stenosis Since the mitral valve is widely opened most of the ventricular filling is completed before auricular contraction so that auricular contraction may produce no murmur A presystolic murmur will therefore usually not be present if the heart rate is slow When the rate is rapid auricular contraction is superimposed in part on rapid ventricular filling and a presystolic murmur is heard (Fig 35) Since similar murmurs may be produced at both the mitral and the tricuspid valve these murmurs are heard over a fairly wide area The point of maximum intensity is commonly just medial to the apex and between the apex and sternum Judged on the basis of intensity these murmurs may be more widely heard than the murmurs of mitral stenosis possibly because of the larger size of the ventricles

The middiastolic murmurs are low pitched with a rumbling quality and are best heard with the bell chest piece in the recumbent or left lateral position When produced at the tricuspid valve they may be louder with inspiration

Occurrence and Differentiation

1 Aortic insufficiency —Since Austin Flint in 1862 described a diastolic murmur at the apex in patients with aortic regurgitation there has been much difference of opinion as to the cause of this phenomenon its frequency and the exact nature of what is heard Considered unusual by some it has been heard frequently

by others the murmur has been described as presystolic middias tolic or both middiastolic and presystolic the murmur has even been considered an auditory illusion Whatever the background may be not infrequently in patients with aortic insufficiency and no mitral involvement a diastolic murmur which to the ear is indis tinguishable from that of mitral stenosis is heard at the apex (Fig 28) There may be a rumbling middiastolic component a presystolic component or both

Much of the controversy regarding the occurence of an Austin Flint murmur has resulted from the fact that the presence of a mid diastolic or presystolic murmur may be simulated by changes in the heart sounds the presence of extra sounds and changes in the early diastolic murmur that occur on transmission to the apex Accentu ated third heart sounds auricular contraction sounds and split first heart sounds might be mistaken for a rumbling middiastolic murmur and presystolic murmur but in most instances do not give the characteristic sound of the murmur of mitral stenosis The early diastolic murmur of aortic insufficiency in transmission to the apex may become lower pitched If careful attention is not paid to timing the murmur may be considered middiastolic especially if the heart rate is somewhat rapid

The apical murmur has been considered to be the murmur of relative mitral stenosis resulting from the enlargement of the left ventricle It has however been reported in hearts showing very little enlargement The rather commonly seen presystolic compo nent is also somewhat unusual in relative mitral stenosis The hy pothesis has been advanced that the anterior cusp of the mitral valve may be partially closed by the regurgitant aortic stream with pro duction of a functional stenosis Another cause may be turbulence produced by the striking together of the mitral inflow stream and the aortic regurgitant stream

Differentiation depends on the history and findings of syphilitic aortitis on one hand and of rheumatic fever on the other If the pa tient has had a *clear-cut episode of rheumatic fever the murmur* should be considered as resulting from mitral stenosis An accentu ated first heart sound is more likely to occur with true mitral steno sis An opening snap of the mitral valve is helpful if present

2 ACUTE RHEUMATIC CARDITIS —A middiastolic rumbling murmur will not infrequently be heard in children in their first at tack of rheumatic carditis Cardiac enlargement is always present

and mitral insufficiency is usually present Since mitral stenosis takes years to develop the occurrence of a middiastolic murmur during the early phases of rheumatic fever would indicate that it was not due to organic mitral stenosis In a child with a large heart and active carditis the diagnosis of organic mitral stenosis should not be made on a middiastolic murmur alone A presystolic component is unusual even when the middiastolic component is loud These murmurs disappear as the inflammation subsides and the heart decreases in size

The differentiation of an organic mitral stenosis and a relative mitral stenosis during an episode of acute rheumatic carditis is often complicated by the fact that it is difficult to be sure whether the attack being witnessed is the first attack or whether there has been scarring of the valves from previous attacks

3 CONGENITAL HEART DISEASE —Apical diastolic murmurs can occur in several forms of congenital heart disease Their occurrence in patent ductus arteriosus and ventricular septal defects causes no difficulty in diagnosis if it is realized that these murmurs may be part of the picture The other characteristic findings usually make the diagnosis apparent The murmur probably results from the increased flow through the mitral valve and the enlargement of the left ventricle

Since atrial septal defects do not have a very characteristic murmur it is important to recognize that apical diastolic murmurs can occur in this condition otherwise the diagnosis of organic mitral *stenosis may be made In most patients considered to have* Lutenbacher s syndrome (atrial septal defect and organic mitral stenosis) the condition has probably been misdiagnosed on this basis The murmur probably results from the increased flow through the tricuspid valve and from the increase in the size of the right ventricle In atrial septal defects the murmur is heard along the left border of the sternum and out toward the apex and consists mainly of a middiastolic component but may at more rapid rates have both middiastolic and presystolic components (Fig 35) Differentiation from mitral stenosis on the basis of auscultation depends on the following (1) a pulmonic systolic murmur and a widely split second sound are usually present (2) the diastolic murmur is better heard between the apex and the lower end of the sternum or along the left border of the sternum (3) no opening snap is present and (4) the first sound is usually not as loud as in mitral stenosis

4 INACTIVE RHEUMATIC HEART DISEASE WITH MARKED MITRAL INSUFFICIENCY —A patient with marked mitral insufficiency and a large left ventricle may have a middiastolic murmur in the ab sence of any mitral stenosis (Fig 29) Patients with marked insuf ficiency and very mild stenosis may have a middiastolic murmur out of all proportion to the degree of the lesion This is especially true in children The murmur results from the increased flow

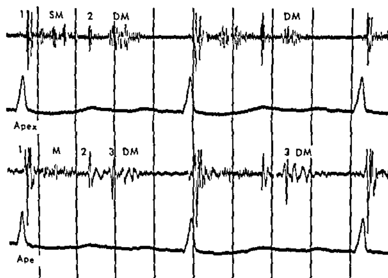


FIG 29 —*Middiastolic murmur in patient with mitral insufficiency* Up per tracing The first sound is followed by a systolic murmur (*SM*) The second sound is of normal intensity A moderately loud middiastolic mur mur (*DM*) is present Lower tracing In a slightly different area at the apex and with a different method of recording it is evident that there is a loud third sound (*3*) followed by a diastolic rumble (*DM*)

through the mitral valve and the enlargement of the left ventricle It may be distinguished from the murmur of organic mitral stenosis on the following bases

a) The presence of a marked mitral insufficiency especially with a systolic expansion of the left auricle indicates that there can not be any important degree of mitral stenosis

b) The murmur at slower rates is nearly always limited to mid diastole even when the murmur is loud and the rhythm regular A presystolic component may occur at rapid rates

c) The first sound is not accentuated

d) An opening snap of the mitral valve is rarely present It is sometimes heard in the presence of a moderate mitral insufficiency associated with moderate stenosis

e) A third heart sound is often present (Fig 29) It may be mistaken for the second heart sound or may be masked by the middiastolic murmur

f) In the presence of a large left ventricle the diagnosis of a significant mitral stenosis must be made with hesitancy even in the presence of a middiastolic murmur

5 LEFT VENTRICULAR ENLARGEMENT —Middiastolic murmurs are occasionally heard in conditions showing only marked left ventricular enlargement The cause of the enlargement may be hypertension coronary artery disease or acute myocarditis other than rheumatic Differentiation is usually not difficult since the murmur is not especially marked and the condition producing the cardiac enlargement evident In cardiac enlargement associated with severe anemia a middiastolic murmur may be heard The increased blood flow associated with the anemia is probably an additional factor in this condition

THE DIASTOLIC MURMUR OF PULMONARY INSUFFICIENCY

(Graham Steell Murmur)

This murmur has the same timing pitch and quality as the murmur of aortic insufficiency The *point of maximum intensity* is in the second or third left intercostal space *Transmission* is down the left border of the sternum but the murmur is not transmitted far unless quite loud Because this murmur is high pitched the *diaphragm* chest piece is preferred Since the pulmonic second sound is accentuated masking occurs it is therefore important to direct attention to the period immediately after the second sound Because pulmonary hypertension is present the second heart sound is usually accentuated What appears to be an accentuated first sound or an early systolic sound may occur (p 38)

*Pulmonary insufficiency is occasionally due to rheumatic fever congenital deformity or an aneurysm but is most commonly asso*ciated with pulmonary hypertension and/or dilation of the pulmonary ring Any condition producing pulmonary hypertension may be associated with this murmur mitral stenosis left heart failure idiopathic or primary pulmonary hypertension pulmonary

hypertension secondary to lung chances such as emphysema and pulmonary hypertension associated with congenital heart diseases

This murmur may be a valuable clue to the patient s clinical condition In patients with pulmonary hypertension associated with lung changes marked variations in the degree of hypertension occur and depend on the status of the lungs In such patients a Graham Steell murmur may come and go depending on the degree of pulmonary hypertension

Differentiation of the murmur of pulmonic insufficiency from that of aortic insufficiency presents difficulties when the peripheral vascular signs of aortic insufficiency are not clear-cut The following factors may help

1 If other evidence of rheumatic heart disease is present it is best to assume that the murmur is that of mild aortic insufficiency unless the evidence in favor of pulmonary hypertension is strong

2 The presence of a systolic murmur of aortic stenosis should usually be considered as evidence that the murmur is due to aortic insufficiency

3 If a loud pulmonic second sound is present and there is no evidence of rheumatic heart disease the murmur may be considered due to pulmonary insufficiency

4 A loud murmur with no peripheral evidence of aortic insufficiency favors the diagnosis of pulmonary insufficiency

5 Wide transmission favors aortic insufficiency The murmur of pulmonary insufficiency is rarely heard in the second right intercostal space

6 In some patients with pulmonary insufficiency the murmur is louder in inspiration than in expiration This respiratory effect is usually not evident and when present it is not as evident as it is in the case of tricuspid murmurs The murmur of aortic insufficiency is usually louder in expiration

7 If the insufficiency is of significant degree fluoroscopy may help by indicating whether it is the aorta or pulmonary artery that shows evidence of increased pulsation A large pulmonary artery favors pulmonary insufficiency

CHAPTER 9

Pericardial Friction Rub, Venous Hum, Extracardiac Auscultation

PERICARDIAL FRICTION RUB

THE PERICARDIAL FRICTION RUB is produced by the rubbing on each other of the parietal and visceral surfaces of the roughened pericardium The sound is usually both systolic and diastolic with a to-and fro character but the systolic component predominates and sometimes the sound is heard only during systole A pericardial friction rub with a clearly recognizable presystolic component is commonly heard postoperatively in patients who have had a mitral commissurotomy and is probably produced by auricular contraction The sound often resembles that produced by squeaky saddle leather and may be described as scratching grating or rasping at times it may be musical The rub seems closer to the ear than the heart sounds especially if some pressure is applied to the chest piece It may be so loud that it masks all other cardiac sounds Occasionally a rub may so closely resemble a murmur that it is difficult to be sure whether a pericardial rub or a murmur is present Continued observation over several days is usually decisive

The rub is most commonly heard between the apex and the sternum but may be very widespread It is usually fairly well localized and persists only for a day or two in cases of myocardial infarction It is much more widespread and may last longer in idiopathic pericarditis and often in rheumatic pericarditis It is heard postoperatively in most cases of cardiac surgery and is loudest in the third or fourth left intercostal space

A characteristic feature of the pericardial rub is its variability The intensity or even the presence of the rub may vary from moment to moment It is markedly influenced by position and may be more evident with the patient in the upright position When looking for a pericardial rub one should check the patient in many dif

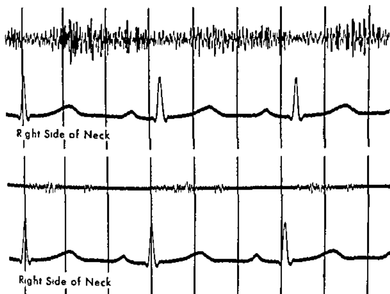


FIG 30—Venous hum This illustrates the same case for which an innocent systolic murmur was shown (Fig 24 *A*) In an attempt to get a tracing from the right side of the neck the patient was instructed to turn her head to the left She did this and raised her chin immediately the loud hum was heard (upper tracing) Note that the hum is loudest during diastole Also note the similarity to the tracing of a murmur of patent ductus (Fig 31) The lower tracing was obtained by placing the finger over the jugular vein above the microphone Nothing else was changed The hum disappeared leaving only a systolic murmur The innocent systolic murmur was somewhat less intense than it was in the left side of the neck (Fig 24 *A*)

ferent positions The intensity of the rub may vary with respiration Often pressure with the stethoscope on the chest wall will increase the rub or make the rub more evident by decreasing the intensity of the heart sounds

VENOUS HUM

The venous hum is a continuous low pitched hum heard in the neck and upper part of the chest in many children and in some

adults (Fig 30) The point of maximum intensity is usually just above the clavicle in the angle between the insertion of the sternocleidomastoid muscle and the clavicle However it is also heard at the base of the heart and rarely may be heard far down the sternum The hum is heard on both the right and the left side but is more common on the right It is better heard with the patient sitting up than with the patient lying down and is accentuated on the right by having the patient turn the head to the left and lift the chin It is louder in diastole Normal respiration may not affect the intensity or may increase it during inspiration

The hum is produced by turbulence in the blood flow in the internal jugular vein possibly where it enters the innominate vein It can therefore be stopped by placing a finger on the internal jugular vein in the neck between the trachea and the sternocleidomastoid muscle at about the level of the thyroid cartilage

The incidence of the hum is increased in conditions associated with increased blood flow e g thyrotoxicosis and anemia

When the hum is well heard at the base of the heart it must be differentiated from the murmur of patent ductus arteriosus The main point in differentiation is to remember the possibility of a venous hum since it is easily recognized if considered and can be stopped by neck pressure

EXTRACARDIAC AUSCULTATION

Auscultation of arteries and veins outside of the thorax may yield helpful information The venous hum that is heard in the neck and may be heard in the precordium has already been described In patients with thyrotoxicosis a continuous bruit may sometimes be heard over the thyroid gland because of a marked increase in vascularity of the gland In some patients with cirrhosis of the liver and portal hypertension a continuous high pitched hum may be heard near the xiphoid and in the epigastric region This venous hum results from the formation of anastomoses between the portal and systemic circulations especially those involving the paraumbilical veins A venous hum may rarely be heard over an enlarged spleen

The continuous murmur heard in a patient with patent ductus arteriosus is the prototype for the murmur heard wherever an arteriovenous fistula or aneurysm exists A continuous murmur with a systolic accentuation and a palpable thrill will occur at the site of the lesion

Normally no murmurs are heard over the arteries However on compression of a larger artery a murmur can be elicited that is synchronous with the pulse wave The murmur is produced by turbulent blood flow at the point of compression In aortic insufficiency and conditions characterized by large pulse pressure and increased blood flow the murmur produced by compression is louder and often both a systolic and a diastolic murmur can be heard This double murmur has been called the sign of Duroziez It is heard in aortic insufficiency and thyrotoxicosis and occasionally in hypertension and in fevers A pistol shot sound over the femoral and larger arteries may be present in aortic insufficiency This sound is loud and short and is produced by the sudden and marked expansion of the almost collapsed artery

CHAPTER 10

Arrhythmias

THE ARRHYTHMIAS ARE NOT covered in this book. Recognition of arrhythmias on auscultation depends primarily on knowledge of the characteristics of the various arrhythmias. In the differential diagnosis of these, auscultation serves merely to give information about rate and timing, both usually easily determined and not depending on special auscultatory skill. The special aid that auscultation can give to the detection of arrhythmias has already been noted (pp. 31-35). Most important is the pathognomonic variation in the intensity of the first heart sound that occurs in complete heart block. A similar variation in the first heart sound in paroxysmal ventricular tachycardia may at times help separate this tachycardia from other forms of tachycardia.

The first sounds of ventricular premature contractions may be more clearly split than those of sinus produced contractions. They are not, however, always split. Auricular premature contractions do not show increased splitting over the regular sinus beats. The first sound of both auricular and ventricular premature beats is often more intense than that of normal beats.

CHAPTER 11

Congenital Heart Disease

THE DISCOVERY OF A cardiac murmur is usually the event that directs attention to the presence of acyanotic congenital heart disease In many acyanotic patients a single defect is present which produces auscultatory findings so characteristic as to be diagnostic In cyanotic patients however several defects are usually present and the auscultatory findings show variations depending on the combination of septal arterial and valvular lesions

Special attention to the character of the second sound yields valuable information Because the deformities are often associated with displacement of the large vessels coming from the heart one cannot judge from location whether a single second sound is coming from the aorta or from the pulmonary artery Careful attention however to the pureness or degree of splitting of the sound will give a valuable clue to whether one or both vessels are present The intensity of the second sound furnishes information on the pressure in the vessels In very small children with rapid heart rates splitting of the second sound is difficult to determine

A systolic murmur that disappears within a week or two may normally be present at birth Persistence of a significant murmur usually indicates some congenital malformation

PATENT DUCTUS ARTERIOSUS

The characteristic murmur of patent ductus arteriosus is a continuous murmur with systolic accentuation (Fig 31) The murmur starts with the first sound increases in intensity to the second sound and then decreases in intensity during diastole reaching a

minimum at the time of the next first sound Many of the murmurs are not actually continuous in that they start a little after the first sound and fade away somewhat before the beginning of the next first sound A thrill can usually be felt when the murmur is loud The intensity of the murmur is increased by exercise Occasionally in an adult at rest the murmur may be so short that it appears to be entirely systolic on exercise a typical continuous murmur develops The intensity of the murmur shows only a rough correlation with the size of the lesion

The characteristic continuous murmur may be present during the first year of life Commonly however only a systolic mur



FIG 31 —Murmur of patent ductus arteriosus This is a continuous murmur which reaches its peak at about the time of the second heart sound and often masks the second heart sound even though the latter may be accentuated It diminishes during diastole

mur can be recognized during the first 6 to 18 months then a continuous murmur becomes evident Some investigators have recently indicated that the presence of only a systolic murmur even during the first year or two of life is indicative of the presence of pulmonary hypertension or cardiac failure This view is not acceptable to those who have followed children for a number of years have heard a systolic murmur change into a typical continuous murmur and have later had the child operated on for an uncomplicated patent ductus It is true that an infant in cardiac failure because of a large patent ductus will often have only a faint systolic murmur

The *point of maximum intensity* of the murmur is almost always in the second left intercostal space next to the sternum occasionally it may be in the first left intercostal space When the murmur is faint it may be limited to this area with increased intensity the systolic component is much better transmitted than the diastolic component and can be heard along the left border of the sternum

and occasionally at the apex (Fig 32) When the murmur is loud anteriorly the systolic component may be heard in the inter scapular region especially in children Transmission to the neck is not especially good

When the murmur is faint it is medium pitched and rumbling as it becomes louder it becomes more harsh The murmur has the same quality during systole and diastole and because of its harshness and its varying intensity during the cardiac cycle it has been described as a machinery type murmur—a very descriptive term The murmur is well heard with either the bell or the dia phragm

Since the murmur is most intense at the time of the second sound this sound is often somewhat masked and may not be obvious even when accentuated The first sound is unchanged

Apical diastolic murmurs similar to those heard in mitral stenosis are frequently heard in children with patent ductus arteriosus The murmur is usually middiastolic but occasionally at rapid rates a presystolic component may be present The occurrence of such a murmur is evidence of a marked shunt The factors in the production of these murmurs have been discussed (p 98) The murmurs disappear with repair of the ductus

Some patients with patent ductus arteriosus develop marked pul monary hypertension This may occur at any age The pulmonary hypertension first decreases the flow through the duct during dias tole the diastolic component of the murmur disappears and only the systolic component remains (Fig 33) With further increase in pulmonary pressure the murmur may disappear or become in significant Some of these patients will develop the early diastolic murmur of pulmonary insufficiency The pulmonic second sound is accentuated An ejection sound or early systolic sound at times may be heard in the pulmonic area The murmur of tricuspid insuf ficiency is commonly present A middiastolic murmur previously present becomes less evident or disappears

Following the successful closure of a patent ductus arteriosus the continuous murmur always disappears and often no murmur at all remains Occasionally a systolic murmur in the pulmonary region may persist even this murmur may disappear over a period of months or years

DIFFERENTIATION —1 When the murmur of a patent ductus ar teriosus in an infant is entirely systolic it is often difficult or impos sible to separate it from the murmur of a ventricular septal defect

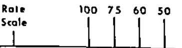


Fig 3? —Murmur of patent ductus arteriosus The figures in parentheses refer to the lines on the chart The following features are shown
a) Murmur is u ually of max mum nten ty in the fir t o econd left int costal pace (7)

b) The murmur is rather harsh It starts with the first sound re ches a ma mum at the time of the second sound and then diminishes during diastole

c) If the murmur is loud the systolic component may be transmitted down along the left border of the sternum (8) and somewhat into the neck (6) and right side of the sternum (2) The murmur is not especially well transmitted to the neck

d) An apical middiastolic murmur that is low pitched and rumbling is often present at the apex (9) (10) if there is a large shunt. This is best heard with the bell and when the patient is in the recumbent or left lateral position

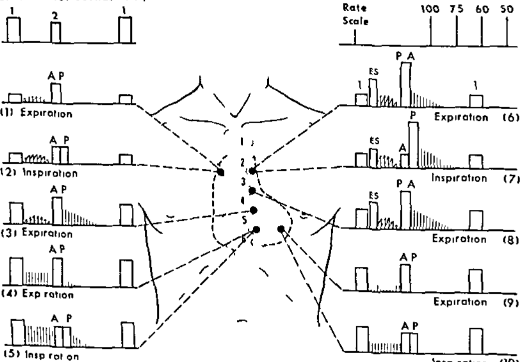


FIG 33.—Patent ductus arteriosus with pulmonary hypertension The following features are shown

a) The continuous murmur previously present disappears and a rough systolic murmur may

pre ent be heard along the left bor ler of the t rnum Somet n e no mu mur at all ı

b) The pulmon c econd sound is markedly accentuated (6) (7) and becau e the pulmo-nary ound is much louder than the aort c second ound splitting is not too evident The pl tt ng may be m re evident lower lown the ternum (4) (5) where the pulmonic second sound is not as loud as it is in the econd and third left intercostal spaces The degree of pl ttıng may be de creased

c) Usually pulmonic insufficiency develops with the production of a high pitched early diastol ıc murmur heard along the left border of the sternum (6) (7) (8)

d) A tricuspid insufficiency often develops This is recognized by a high pitched systolic mur mur of maximum intensity at the lower end of the sternum This murmur is louder in inspiration (5) than it is in expiration (4)

e) Frequently an early systolic sound (*ES*) is heard in the second and third left intercostal spaces This is of clicking quality and louder in expiration (6) than in inspiration (7)

The heart is small and the difference in point of maximum intensity is not great enough to be of importance

2 A venous hum can be confusing only if one does not consider the possibility of its presence The hum will be loudest above the clavicle usually on the right and can be stopped by correct pressure on the neck

3 An aortopulmonary septal defect a defect between the aorta and pulmonary artery just above their origin gives a murmur almost identical with that of patent ductus arteriosus in timing and quality In this condition the point of maximum intensity of the murmur may be in the third intercostal space and may be somewhat more medial than that of patent ductus arteriosus The murmur is usually loud and harsh Perforation of an aortic sinus into the pulmonary artery will give a similar murmur

4 In a tetralogy with severe stenosis or atresia of the pulmonary artery and no patent ductus arteriosus the blood reaches the lungs through markedly dilated and tortuous bronchial arteries In such a condition one may hear a continuous murmur very similar in quality to that of patent ductus arteriosus The murmur is much more diffusely heard however than that of patent ductus arteriosus and is usually better heard in the back The murmur is apparently produced by vibrations of the tortuous bronchial arteries A pure second sound is heard in this condition

5 The murmur that occurs after a successful shunt operation for congenital heart disease is also similar to that of patent ductus arteriosus The location will vary with the location of the shunt

6 Other conditions producing a continuous murmur include (*a*) perforation of an aortic sinus into the right ventricle The maximum intensity of the murmur is lower than in patent ductus arteriosus and may be in the fourth left intercostal space (*b*) arteriovenous aneurysms of any of the vessels in the thorax—bronchial pulmonary coronary or intercostal (*c*) total anomalous drainage of the pulmonary veins

VENTRICULAR SEPTAL DEFECTS

The murmur of a ventricular septal defect is systolic (Fig 34) it extends throughout systole and when loud has a peak in mid systole (diamond shaped) Less intense murmurs may be maximum earlier in systole The murmur consists of high and medium frequency vibrations and when of any intensity it is quite harsh A

thrill is present with louder murmurs The murmur may be very loud The point of maximum intensity is most often in the third or fourth intercostal space to the left of the sternum Rarely very high septal defects give a murmur that is most intense in the second left intercostal space when the patient is lying down Transmission

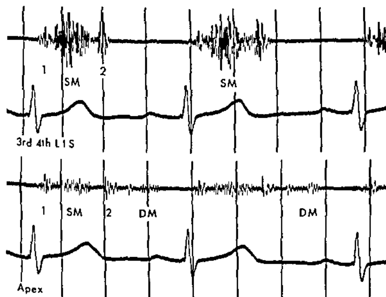


FIG 34—Ventricular septal defect Upper tracing A loud systolic murmur (SM) with a diamond shape is present in the third to fourth left intercostal spaces It extends to the second sound but the second sound is evident Lower tracing A middiastolic murmur is evident at the apex This is often present with large shunts and indicates a relative mitral stenosis The systolic murmur is much less intense at the apex than along the left border of the sternum

depends mainly on the intensity the more intense murmurs may be heard over the entire precordium and posteriorly but are poorly heard in the neck

The heart sounds are unchanged when the defect is not marked Loud murmurs may partially mask the aortic second sound When the defect is severe the pulmonic second sound may be accentuated Splitting of the aortic and pulmonic components of the second sound is of normal degree

When the defect causes left ventricular enlargement a rumbling apical middiastolic murmur may be present (Fig 34) An occa

sional patient with a very high ventricular septal defect will have an incompetent medial cusp of the aortic valve which gives an early diastolic murmur, the combination of systolic and early diastolic murmurs may be mistaken for the continuous murmur of a patent ductus arteriosus

If pulmonary hypertension develops the left to right shunt is decreased and the murmur becomes less intense and less harsh With a mixed shunt the murmur may become quite faint Under such conditions the pulmonic second sound is increased in loudness pulmonary insufficiency commonly occurs and a tricuspid insufficiency develops A pulmonary ejection sound may occur The auscultatory findings may now be indistinguishable from those of patent ductus arteriosus with pulmonary hypertension (Fig 33)

Innocent systolic murmurs that are most intense in the third and fourth intercostal space may at times be difficult to distinguish from the murmur of a small ventricular septal defect (maladie de Roger) In general the murmur of a small ventricular septal defect is more localized and of different quality than the innocent murmur (p 86)

The murmur of mild pulmonary valvular stenosis usually has a point of maximum intensity higher than that of a ventricular septal defect and if the second sound can be heard it is more widely split (Fig 38) A mild isolated pulmonary infundibular stenosis however may give a murmur very similar in quality and location and if increased splitting of the second sound cannot be determined auscultatory differentiation may be impossible

The auscultatory findings in *Eisenmenger s complex* are those of a ventricular septal defect with pulmonary hypertension

ATRIAL SEPTAL DEFECT

Even in its milder form an atrial septal defect is almost always associated with a systolic murmur and a widely split second sound (Figs 35 and 38) The systolic murmur is of medium pitch and rarely as harsh or loud as that of a ventricular septal defect A thrill rarely occurs The point of maximum intensity is usually in the second but may be in the third left intercostal space and the murmur is often transmitted to the left along the second intercostal space The murmur may be due to a relative pulmonary stenosis resulting from enlargement of the right ventricle and pulmonary

artery and from the marked increase in blood flow through the valve

The second sound is almost always widely split. The pulmonic component is not usually accentuated but in some patients may show mild to moderate accentuation. The pulmonic second sound although of normal intensity is more widely heard than usual and

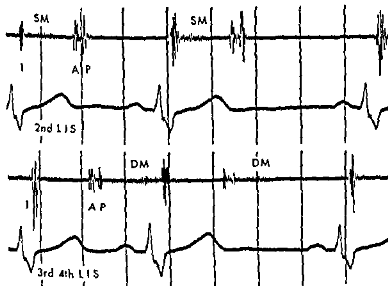


Fig 35.—Atrial septal defect. Upper tracing. The systolic murmur (*SM*) in the second left interspace is of medium pitch and not very loud. It sounds much like an innocent murmur that is of maximum intensity in the second left intercostal space. The second heart sound is clearly split in both inspiration and expiration. Lower tracing. In the third to fourth left intercostal spaces a middiastolic murmur (*DM*) is present. Although one does not expect to get a presystolic murmur in a functional mitral or tricuspid stenosis this tracing shows why a presystolic murmur may occur. Following the second beat there is a long diastole; one sees only a middiastolic murmur with no presystolic murmur. However, after the first beat diastole is shortened by the more rapid rate; atrial contraction now falls during or near the period of rapid ventricular filling and the result is a combination of a middiastolic and a presystolic murmur.

the split second sound is therefore evident along the left border of the sternum and often at the apex (Fig 15). With large atrial septal defects an increase in the splitting of the second sound with inspiration may not be evident; presumably the flow into the right auricle as a result of the defect is so great that inspiration does not further increase the filling of the right auricle and ventricle. The first sound may be somewhat increased in loudness.

A rumbling middiastolic murmur is not infrequently heard between the apex and the left border of the sternum or along the left border of the sternum (Fig 35) This murmur may be produced by a marked increase in flow through the tricuspid valve the increase in flow producing a relative tricuspid stenosis At times an auricular sound may be heard in the same area (Fig 2 D) The mid diastolic murmur and the auricular sound may increase in loudness with inspiration

If pulmonary hypertension develops the following changes occur (1) the pulmonic second sound increases in loudness (2) an early diastolic murmur of pulmonary insufficiency frequently develops, (3) a pulmonic early systolic ejection sound may be heard (4) with right ventricular failure the systolic murmur of tricuspid insufficiency frequently is present and may be loud (5) if the pulmonary hypertension is severe the degree of splitting of the second sound is decreased and may be of normal degree or entirely absent

It is obvious that the auscultatory findings in atrial septal defect with severe hypertension are similar to those already described for patent ductus arteriosus and ventricular septal defect with pulmonary hypertension (Fig 33) Similar auscultatory findings may occur in *primary pulmonary hypertension*

The pulmonic early systolic sound may occasionally occur in the absence of pulmonary hypertension

The auscultatory findings in *partial anomalous pulmonary venous drainage* are the same as in atrial septal defect In some patients with a persistent *ostium primum* a deformed mitral valve produces the murmur of mitral insufficiency and helps to differentiate this lesion from defects in other parts of the septum

COARCTATION OF THE AORTA

Although coarctation of the aorta sometimes produces no auscultatory findings it usually causes a basal systolic murmur which is heard as well or better posteriorly in the interscapular region Anteriorly the systolic murmur is of maximum intensity usually in the second left intercostal space but occasionally on the right and rarely at the apex It may be difficult to decide whether the murmur is louder on the left or the right but this in itself is significant indicating the deep origin of the murmur Over the dorsal spine the murmur may be continuous and not infrequently is both systolic

and diastolic although it is difficult to recognize this since the heart sounds are not well heard The diastolic element may be recognized by using the symballophone The murmur is rarely loud anteriorly and the presence of a loud murmur should make one consider the possibility of additional defects

The important feature of the murmur of coarctation is not that it is well heard in the interscapular region since many loud murmurs may be heard there but that it is often louder in the interscapular region than it is anteriorly

Sometimes the early diastolic murmur of aortic insufficiency is present and suggests a bicuspid aortic valve Because of the increased systemic pressure the aortic second sound may be increased Occasionally systolic murmurs are heard along the left border of the sternum These may represent associated anomalies since most of them persist after operation

AORTIC AND SUBAORTIC STENOSIS

The auscultatory findings in congenital aortic stenosis are those described for aortic stenosis (p 76) The systolic murmur of subaortic stenosis has the same pitch and quality as that of aortic stenosis Differentiation of aortic and subaortic stenosis is difficult and usually not possible on the basis of auscultation Theoretically the point of maximum intensity should be somewhat lower in subaortic stenosis than in aortic stenosis and a maximum intensity in the third left intercostal space should suggest subaortic stenosis The aortic second sound is usually of normal loudness in subaortic stenosis but may be diminished in aortic stenosis however a normal aortic second sound is usually present in mild aortic stenosis and at times in severe aortic stenosis Tracings of the carotid pulse should show an incisura followed by after vibrations in subaortic stenosis and no incisura in aortic stenosis but I have found this point to be of little value since both tracings can be obtained in aortic stenosis An early diastolic murmur of aortic insufficiency suggests valvular rather than subvalvular stenosis

PULMONARY STENOSIS WITH AN INTACT VENTRICULAR SEPTUM

Pulmonary stenosis with an intact ventricular septum is associated with a harsh systolic murmur of maximum intensity in the second left intercostal space (Figs 36 and 38) The murmur has

much the same pitch and quality as that of aortic stenosis and on a phonocardiogram is also diamond shaped There is a poor correla tion between the intensity of the murmur and the degree of stenosis

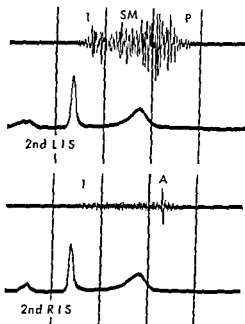


FIG 36.—Pulmonary stenosis with an intact ventricular septum The *first heart sound is faint and followed by a long systolic murmur* which reaches a peak very late and ends with a faint pulmonic second sound The second sound is not evident on auscultation In the second right intercostal space the murmur is much less intense and the aortic second sound is heard before the end of the murmur It is evident that in the second left inter costal space the aortic second sound is masked by the murmur

The murmur is often widely transmitted and is better heard on the left side of the neck than on the right A thrill is usually present if the murmur is loud

In severe pulmonary stenosis with an intact ventricular septum right ventricular systole is prolonged because of the increased load and delayed emptying This results in a long murmur that extends through and masks the second aortic sound (FIG 36) Since the pulmonic second sound is faint or absent no second sound is evident in the area where the murmur is loudest (Fig 38) although an aortic second sound may be heard at the apex and in the second right intercostal space With milder degrees of pulmonary stenosis

the pulmonic second sound is usually present and may be of normal intensity Even with mild stenosis however the pulmonic second sound is nearly always delayed and wide splitting is present If the murmur is not too loud this splitting is usually very noticeable The splitting is often more evident in the third left intercostal space where the murmur is not so loud and the sounds better heard

In mild pulmonary stenosis an early systolic sound (p 38) may occasionally be heard in the second and third left intercostal spaces An auricular sound may occur resulting in a presystolic triple rhythm The hypertrophy of the right auricle may be a factor in its production

TETRALOGY OF FALLOT

Tetralogy of Fallot is usually associated with a systolic murmur heard along the left border of the sternum (Figs 37 and 38) With very severe grades of pulmonary stenosis or with pulmonary atresia there may be no murmur and it would seem that the murmur when present is primarily due to the pulmonary stenosis Since in fundibular stenosis is most common in tetralogy of Fallot the murmur is usually loudest in the third or fourth left intercostal space The murmur is harsh and often loud but in general it is not as loud as the murmur of pulmonic valvular stenosis with intact

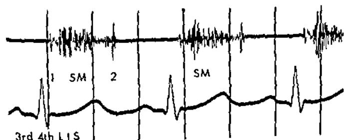


FIG 37 —Tetralogy of Fallot The systolic murmur (*SM*) is faint by the time the second sound occurs The second sound is clearly heard and shows no evidence of splitting The second sound is produced by the aortic valve

ventricular septum Whereas in pure pulmonary stenosis the right ventricle can empty itself only through a stenosed pulmonic valve in tetralogy the right ventricle can easily empty itself into the aorta through the ventricular septal defect Right ventricular systole is therefore not prolonged and the murmur produced is shorter th

2nd L I S 3rd L I S 4th L I S













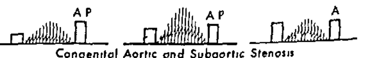


FIG 38.—Comparison of murmurs in more common types of congenital heart disease The murmurs and heart sounds are shown in the second third and fourth left intercostal spaces The second heart sounds are all shown in *inspiration* For changes in the second heart sound with respiration in these conditions see Figure 12 The point of maximum intensity of the murmur is shown by the height of the lines indicating the murmur and by underscoring the base line The murmur of congenital aortic stenosis is maximum in the second right intercostal space (not shown)

that in pure pulmonic stenosis The systolic murmur in tetralogy stops somewhat before the second sound permitting the second sound to be well heard

The second sound in tetralogy tends to be pure since the pulmonic second sound is faint and not usually heard Because of displacement of the aorta the second sound that is heard to the left of the sternum is the aortic second sound At the site where the murmur is loudest the second sound is thus often clearly heard in contrast to what is found in pulmonary stenosis with an intact ventricular septum

OTHER CONGENITAL HEART LESIONS

In *transposition of the great vessels* a murmur is usually associated with the presence of a ventricular septal defect When this defect is not present there may be no murmur The second sound is often booming and two components are present but are not often easy to recognize because of the rapid heart rate

In *tricuspid atresia* the murmur may be due to the presence of a ventricular septal defect Occasionally the murmur of mitral insufficiency is heard

In *Ebstein s disease* a wide variety of findings may occur A widely split second sound is common because of the frequent presence of some right bundle branch block A systolic murmur may be present and is probably due to tricuspid insufficiency but it is often more harsh than the usual tricuspid insufficiency murmur A middiastolic and occasionally a presystolic murmur probably associated with the tricuspid deformity may occur The murmurs may seem to be quite loud at the apex but it should be recalled that in this condition the tricuspid valve is usually far to the left A triple rhythm probably due to an auricular sound may at times be heard along the left border of the sternum

CHAPTER 12

Auscultatory Phenomena in Rheumatic Heart Disease

ACUTE RHEUMATIC CARDITIS

1. The first sound may be diminished because of prolongation of atrioventricular conduction (p. 33).

2. Wenckebach's phenomenon (recognized by recurrent dropped beats) and a 2:1 block occur with more severe involvement of the conduction system.

3. A pericardial rub may be present early.

4. A systolic murmur with the characteristics of an innocent murmur is often heard (p. 82). This is not due to valvular involvement and may be associated with the fever and increased heart action.

5. A gallop rhythm may develop if the heart enlarges and failure occurs (p. 57).

6. The faint murmur of mitral insufficiency (p. 68) may be present early and remain faint or increase rapidly in loudness. If the heart size is normal, the murmur is produced by valvular deformity resulting from the acute endocarditis. Cardiac enlargement is associated with a loud systolic murmur that is usually due in part to relative mitral insufficiency.

7. A middiastolic murmur that does not indicate organic mitral stenosis may occur with the cardiac enlargement (p. 99).

8. A faint murmur of aortic insufficiency (p. 93) is sometimes present early, often with no other murmur.

9. A pulmonary systolic murmur, rather musical in quality

and often varying from day to day occasionally occurs Its cause and significance are not clear

REGRESSION OF THE ACUTE PHASE —1 The first heart sound improves as the atrioventricular conduction time shortens

2 The pericardial rub disappears

3 The middiastolic murmur disappears as the heart decreases in size

4 The systolic murmur may become much less intense and if not too loud when the heart was enlarged it may disappear entirely or become very faint

5 The murmur of aortic insufficiency usually persists but may if it was faint disappear If associated with peripheral signs of insufficiency it does not disappear

SUBSEQUENT COURSE —The subsequent course depends on the amount of inflammation that occurred in the acute phase and on the amount of scarring that will supervene Even more important may be recurrences of activity of rheumatic fever *Rheumatic fever would seem to be a chronic smoldering disease with recurrent flare ups of subacute and subclinical severity in addition to the recogni ed acute incidents Repeated insults to the valve over a period of years rather than one injury with scarring account for the damage*

MITRAL VALVE INVOLVEMENT

Figure 39 presents in diagrammatic form the changes that can occur in the mitral valve As a result of an episode of acute rheumatic fever the patient may be left with a mild mitral insufficiency The mitral valve cusps are somewhat thickened and possibly shortened but remain flexible The edges of the cusps may be firm and rolled The chordae tendineae remain flexible but may also be somewhat thickened and shortened The mitral insufficiency produces a typical murmur This murmur can be recognized and the diagnosis of mitral insufficiency can be made in the absence of any other finding The heart sounds are essentially unchanged Occasionally a patient may recover from acute rheumatic fever with no evidence of valvular damage and then over a period of years with no evidence of further rheumatic activity develop a murmur of mild mitral insufficiency

The involvement of the mitral valve may stop with production of a mild or moderate insufficiency which can be recognized only by

NORMAL MITRAL VALVE

Ep od of
h mat f

M'ld M t I I ff y
C ps omewhat th k d d
horte ed b t t ll fl bl R lled
dges. Mode ate th k g f
chord e t d ea

U u lly
s a d
pe ted
p od of
h mat
fe

Us lly m ld d
fte u og ed
ep sod s of
h mat
fe r

R peat d ep sod s
of he mat f

Oft w th obv p od
of h m t c f

Sev M t I I fficiency
(Figur 40)
Thick ing a d hort g f th
cusps and hord t d eae
E larged atr ove tric l ng
Large left ventricle

C mb d M tral St d
I ff cy
(F g 41)
Mod t d g of e h al
though on or th m y be
q e e C p a d chord e
t d o ma kedly f b sed a d
m ti I m t d may b
gid Som k f t on

T ght M t I St s with I ght
m ld M t I I ff cy
C p d hord e t d
th k d b t t ll fl bl Cusp
dg oll d d th ck ed
C mm f sed

T ght M t I St o
(F g 42)
No m t I ff cy Comm s
ur s f sed C ps a d horda
t d ne th ck ned b t t ll
fl bl

FIG 39 —Changes in mitral valve resulting from rheumatic fever

the presence of the typical murmur Frequently the involvement progresses even without evidence of acute rheumatic fever Depending on the type and severity of the valvular changes severe mitral insufficiency mitral stenosis or any combination of the two may result Patients with no recognizable recurrences of acute rheumatic fever are more likely to develop mitral stenosis Patients with repeated severe attacks of rheumatic fever are more likely to show a combination of mitral stenosis and mitral insufficiency or severe mitral insufficiency

SEVERE MITRAL INSUFFICIENCY —When *severe mitral insufficiency* with little or no stenosis occurs (Fig 40) the valve cusps become thickened and shortened but retain some flexibility the chordae tendineae are also thickened and may show marked shortening. For some reason the atrioventricular ring is not involved in the fibrosis and seems to enlarge Enlargement of the left ventricle as a result of the mitral insufficiency aggravates the mitral insufficiency because of relative shortening of the chordae tendineae The result is a severe mitral insufficiency with little or no stenosis The auscultatory findings are as follows

1 The first sound is of normal or decreased intensity A loud murmur may mask the sound which may be seen on a phonocardiogram

2 A loud high pitched apical systolic murmur of mitral insufficiency is present (p 68)

3 The second pulmonic sound may be increased if there is an increase in left auricular and pulmonary artery pressure The degree of splitting is usually unchanged but may be increased

4 A third heart sound is often present at the apex and may be louder than the second sound in that area

5 A middiastolic murmur is often heard This is usually of faint or moderate intensity but in children it may be loud A presystolic murmur is not usually present but may occur Differentiation from the murmur of organic mitral stenosis is important (p 101)

COMBINED MITRAL STENOSIS AND INSUFFICIENCY —With extensive involvement of the cusps and the chordae tendineae a *combination of mitral stenosis and mitral insufficiency* results (Fig 41) The cusps and the chordae tendineae become fibrosed and rigid and there is little if any movement of the cusps With severe involvement the valve ceases to function as a valve and there is merely a funnel shaped opening which is usually somewhat larger than that of a tight mitral stenosis During ventricular systole free



Fig 40 —Severe mitral insufficiency The numbers in parentheses refer to the l nes on the chart The following features are shown

a) A lou d high p tched y tolic murmur of maximum nten ity at the apex (9) (10) Th mur

mur is well transmitted into the ax lla (8) and is also heard medially often ι p to the ternal bo der (3) (4) (5) In this area the murmur is louder in exp ration (5) than in in p rat on (4) This murmur is heard in the aortic area only when very loud

/) The first heart sound is diminished in intensity and masked by the loud systolic murmur (8) (9) (10)

c) There is often some splitting of the second sound even in expiration in the second left intercostal space (6) This is increased in inspiration (7)

f) A third heart sound which is often louder than the second heart sound may be present at the apex (9) (10)

e) A middiastolic murmur often follows the third heart sound (10) and in some areas only the middiastolic murmur without the third heart sound may be heard

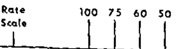


Fig 41—Combined mitral stensosis and mitral insufficiency The following features are il lu trated

a) A sy tol c mu mur of mode at ly loud or loud nten ty at the apex (8) (9 (10)

b) A midd a tol c and p e y t lic rumbl ng mu n Th s loude t w in the pat ent in the left lateral posit on in an area somewhat above the apex and w th the bell nd p ec (8) The ystol c murmur also is loudest with the patient in the left lateral po t on bt t usually n an area below that at which the diastolic murmur is loudest—that is at the apex or sl ghtly below the apex (10) It is best heard with the diaphragm

c) A normal or diminished first heart sound It is occasionally accentuated

f) A normal splitting of the second heart sound (6) (7)

regurgitation occurs but is limited by the size of the opening There will be a variation in the size of the opening and a corresponding reciprocal relationship between the amounts of stenosis and of insufficiency Calcium deposits occur frequently The involvement of the valve is often not uniform and one commissure or cusp may be more involved than the other The auscultatory findings are as follows

1 The first sound is diminished

2 A moderately loud or loud systolic murmur of mitral insufficiency is present

3 *An accentuated pulmonic second sound may occur if the pul*monary arterial pressure is increased The splitting of the second sound is normal

4 The middiastolic and presystolic murmurs of mitral stenosis are moderately loud or loud With mitral valve openings of 1½ to 2 sq cm both the systolic and the diastolic murmurs may be loud

5 Neither an opening snap of the mitral valve nor a third heart sound is present

TIGHT MITRAL STENOSIS —In *tight mitral stenosis* (Fig 42 *A* and *B*) without any insufficiency or with minimal mitral insufficiency the pathologic change is primarily a fusion of the commissures of the valve The cusps and chordae tendineae remain flexible and only mildly fibrosed The edge of the cusp is rolled and thickened Many patients with tight mitral stenosis have no history of acute rheumatic fever The auscultatory findings are as follows

1 The first sound is accentuated

2 The middiastolic and presystolic murmurs are moderately loud or loud

3 An opening snap of the mitral valve is present

4 A faint or moderately loud systolic murmur of mitral insufficiency may be present but frequently no systolic murmur is heard

5 The second sound is normal and shows normal splitting What is often described as a splitting of the second sound is usually due to the presence of an opening snap of the mitral valve The pulmonic second sound may become accentuated with increase in pulmonary arterial pressure

With the development of auricular fibrillation the presystolic murmur of mitral stenosis if present disappears The findings are otherwise essentially unchanged

TIGHT MITRAL STENOSIS WITH EARLY PULMONARY HYPERTENSION —When severe mitral involvement is present for a number

of years pulmonary hypertension right ventricular hypertrophy and dilation and tricuspid insufficiency occur These produce the following auscultatory changes (Fig 43)

1 The pulmonic second sound is accentuated

2 An early diastolic murmur of pulmonary insufficiency (Graham Steell murmur) may develop

3 The systolic murmur of tricuspid insufficiency is heard This

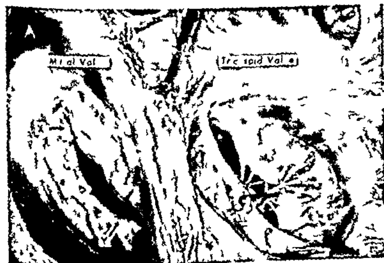


FIG 43 *A* —Tight mitral stenosis and mild organic tricuspid insufficiency Autopsy specimen from a 30 year-old woman who died of an arrhythmia 8 e hours after a mitral commissurotomy The a ria have been cut away to expose both valves The mitral valve was thickened but was quite flexible The surgeon had fractured the valve along the anterolateral commissure (X) and along the posteromedial commissure (Y) The valve opening was originally about 9 mm long (the dark area in the center) The tricuspid valve shows a thickening and rolling of the valve edg that a counted for th murmur of tricuspid insufficiency

can be recognized by its location and its increased intensity with inspiration (p 74)

TIGHT MITRAL STENOSIS WITH PULMONARY HYPERTENSION AND SEVERE TRICUSPID INSUFFICIENCY —The auscultatory findings in tight mitral stenosis may be so changed by marked tricuspid insufficiency and right ventricular enlargement that the diagnosis of mitral stenosis is made with difficulty The right ventricle occupies most of the anterior precordium so that the auscultatory evidence of mitral stenosis is shifted to the axillary region In addition a decreased

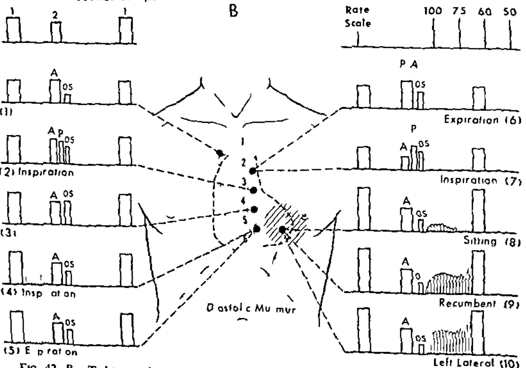


FIG 42 B—Tight mitral stenosis with flexible valve and no mitral insufficiency A very mild tricu pid insufficiency is present These are the auscultatory findings for the patient whose heart valves are shown in Figure 42 A The following typical features are shown

a) An accentuated first sound. This is most evident at the apex (8) (9) (10) but its intensity of sound is increased in other areas also.

b) A middiastolic and presystolic murmur at the apex (8) (9) (10). This murmur is quite faint when the patient is in the sitting position (8) and much better heard when she is recumbent (9). In the left lateral position the murmur is still further accentuated (10).

c) An opening snap of the mitral valve. This is loudest and most evident in the fourth left intercostal space (3) but is heard over the entire precordium. In the second left intercostal space it is most evident in expiration (6). In inspiration (7) it follows so closely upon the pulmonic second sound that it is heard with difficulty.

d) A normally split second heart sound. The second sound is split in the second and third left interspaces in inspiration (7). There is no split in expiration (6). This amount of splitting is within normal limits.

e) The murmur of tricuspid insufficiency. The murmur heard to the left of the lower end of the sternum is high pitched and best heard with the diaphragm (4) (5). It shows a definite increase in loudness in inspiration.

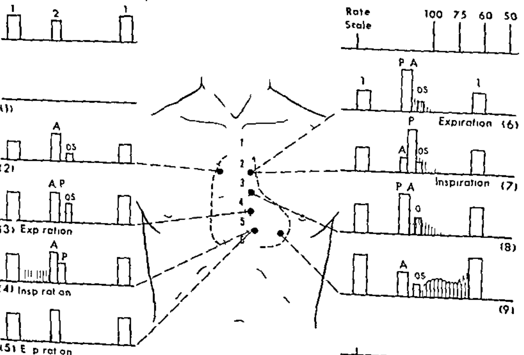


FIG 43.—Tight mitral stenosis with early pulmonary hypertension The following features are shown

a) An accentuated fir t heart sound

b) A *pulmonic second heart sound accentuated* as a result of the *pulmonary hypertension* The degree of splitting of the *second sound* is normal Because the *pulmonic* component is accentuated the *splitting of the second sound* is evident over a *wider area than normal* (4)

c) *An opening snap of the mitral valve*

d) The middiastolic and presystolic murmurs of *mitral stenosis* (9)

e) A high pitched early diastolic murmur in the second and third left intercostal spaces (6) (7) (8) This is the *murmur of pulmonary insufficiency* It may at times be more evident in inspiration (7) than in expiration (6)

f) Some tricuspid insufficiency It is indicated by a *high pitched systolic murmur heard best at the lower end of the sternum louder in inspiration* (4) than in expiration (5)

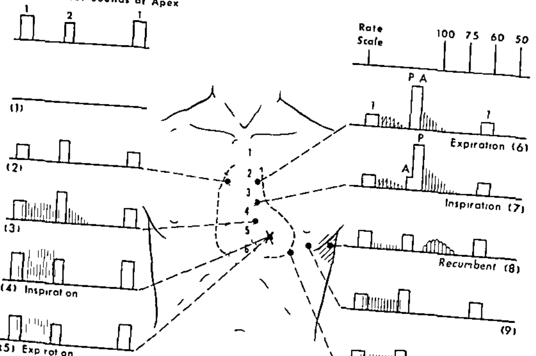


Fig 44.—Tight mitral stenosis with pulmonary hypertension and severe tricuspid insufficiency The following features are shown

a) A loud high pitched systolic murmur dominates the auscultatory findings This is of maxi

mum intensity at the lower end of the sternum or between the ste num and the pex It h d in diminishing loudness from its point of maximum intens ty and may be heard fa rly w ll t the apex (10) and even more laterally (9) Because of the loudne s of this murmur changes in intensity with respiration may not be evident

b) The murmur usually masks the first heart sound

c) The evidences of mitral stenosis are shifted laterally to the anterior axillary and even mid axillary region (shaded area) Here (8) the systolic murmur is faint and a rumbling diastolic murmur may be evident It is not usually very loud

d) The pulmonic second sound is accentuated and the degree of splitting may be somewhat diminished (6) (7)

e) A high pitched early diastolic murmur is heard along the left border of the sternum (3) (6) (7) This is the murmur of pulmonic insufficiency

cardiac output results in a faint middiastolic murmur The evidences of tricuspid insufficiency are dominant (Fig 44)

1 The first sound is normal or somewhat diminished but often masked by a loud murmur of tricuspid insufficiency

2 A loud systolic murmur is heard over most of the precordium but is usually recognizable as having its origin in the tricuspid valve by the following findings

a) The maximum intensity is at the lower end of the sternum or somewhat more lateral but usually within the midclavicular line This murmur fades laterally more rapidly than would a loud murmur due to mitral insufficiency in a large heart

b) The murmur usually increases with inspiration (p 74)

3 As one moves the stethoscope laterally over the chest the systolic murmur fades and one hears a clear but usually rather faint first heart sound in an area where the systolic murmur is faint or absent This may be at the anterior or at the midaxillary line

4 In this area where the first heart sound is heard a faint mid diastolic murmur is usually evident Occasionally no diastolic murmur can be heard

5 The murmur of pulmonary insufficiency is usually present

If the condition is compensated by intensive therapy the auscultatory pattern gradually shifts back to that of a mild tricuspid insufficiency with recognizable mitral involvement (Fig 43) sometimes the evidence of tricuspid insufficiency disappears almost completely leaving only the classic evidences of mitral stenosis (Fig 42 *B*) The Graham Steell murmur and the accentuated pulmonic second sound often persist even if the tricuspid insufficiency murmur disappears

It is extremely important to recognize patients with tight mitral stenosis in whom the tricuspid insufficiency dominates the auscultatory findings because these patients are often excellent candidates for mitral commissurotomy

AORTIC VALVE INVOLVEMENT

Aortic valve involvement occurs most commonly in association with mitral valve involvement but may be isolated A faint early diastolic murmur of aortic regurgitation occurs sometimes early in the course of acute rheumatic carditis This murmur usually persists but it frequently goes unrecognized because it is difficult to hear There may be no associated systolic murmur in the aortic

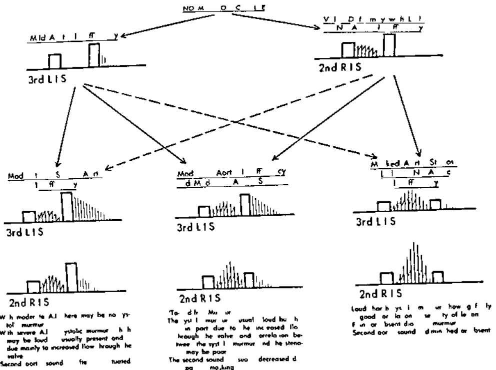


FIG 45—Changes in aortic valve resulting from rheumatic fever

area Mild to moderate degrees of aortic regurgitation may develop fairly soon after the acute episode (Fig 45) When the murmur of aortic insufficiency is moderately loud or loud there is almost always an associated systolic murmur in the second right intercostal space The systolic murmur may be produced by deformity of the valve An increased velocity of blood flow through the valve during systole can probably in itself produce the murmur or accentuate the murmur produced by any valvular deformity The second heart *sound may be accentuated*

A more common result than severe aortic insufficiency with little or no stenosis is the combination of insufficiency and stenosis (Fig 45) This condition will give a loud systolic murmur and moderately loud to loud diastolic murmur The systolic murmur is often out of proportion to the degree of stenosis again because of the increased flow due to the regurgitation The aortic second sound is decreased The systolic murmur is well heard at the apex and often masks the murmur of mitral insufficiency

Severe degrees of aortic stenosis with little or no regurgitation may possibly result from a continuation of the process that produces a moderate stenosis and regurgitation However if calcific aortic stenosis is of rheumatic origin it is obvious that aortic stenosis can result without a stage during which there is significant aortic regurgitation With severe aortic stenosis such as occurs with calcific aortic stenosis the systolic murmur may be very loud and a diastolic murmur of insufficiency is often not heard and if it is heard may be quite faint The aortic second sound is faint or absent at the base but often when not heard at the base it may be heard at the apex

During acute rheumatic fever a systolic murmur of medium pitch may develop in the aortic region which may or may not be associated with a faint early diastolic murmur This systolic murmur may persist unchanged for years or become louder and then usually becomes associated with some early diastolic murmur When of moderate intensity it may not have the harshness of a murmur of aortic stenosis and if no history of rheumatic fever is obtained its significance may be difficult to determine It is always important in this instance to make a careful search for a murmur of aortic insufficiency even though it be of the faintest degree because such a murmur will immediately make the diagnosis

When loud systolic and diastolic murmurs are present in aortic valvular disease they are often described as being 'to-and fro'

This term should not be used for the continuous murmur of patent ductus arteriosus

A middiastolic or presystolic murmur associated with aortic in sufficiency in a patient with rheumatic heart disease should be con sidered as resulting from organic mitral involvement rather than relative mitral stenosis

SELECTION OF PATIENTS WITH VALVULAR INVOLVEMENT FOR CARDIAC SURGERY

MITRAL COMMISSUROTOMY —The ideal patient for mitral com missurotomy has a significant or tight mitral stenosis and a valve flexible enough to act after the operation very much like a normal valve Such a patient shows the auscultatory findings illustrated in Figure 42 *B* The long middiastolic murmur the presystolic mur mur and the absent or faint systolic murmur indicate the presence of a tight mitral stenosis The loud first sound and the opening snap of the mitral valve are evidence that the valve is fairly flexible The result of mitral commissurotomy in such a patient is dramatic and most satisfying

Until a successful operation is available for mitral insufficiency the patient with a marked mitral insufficiency and minimal stenosis should not be operated upon with the mistaken idea that stenosis is the predominant lesion The recognition of this patient from the auscultatory standpoint has been described (Fig 40 and p 129)

Patients who have pronounced pathology of the valve with a combination of stenosis and insufficiency represent a difficult group It is obvious that if a valve and the chordae tendineae are markedly fibrosed and bound down commissurotomy will be of little avail since there will be nothing to hold the valves apart The commissures often cannot be recognized and little can be done because of calci fication and fibrosis Fortunately in many of these patients the pathology is spotty and although one commissure may be markedly involved the other can be opened successfully with some benefit to the patient If the stenosis appears to be the main lesion and if calci fication is not pronounced many of these patients deserve a chance to be helped by an operation This is true especially if they are going downhill rapidly Improvement is occasionally striking and many may receive some benefit Pulmonary hypertension is common in these patients and when severe is a contraindication to operation

Patients whose condition *must* be recognized are those in whom

the signs of a tight mitral stenosis are masked by the presence of tricuspid insufficiency In such patients operation often gives dramatic results The operation however must be done before the pulmonary hypertension becomes fixed and irreversible

EFFECT OF MITRAL COMMISSUROTOMY ON MITRAL MURMURS — During the first week following commissurotomy all murmurs may be faint or absent Thereafter a gradual return of the diastolic murmur occurs The diastolic murmur rarely disappears completely even when the results are excellent the murmur may be faint but it can usually be heard by listening to the patient in the left lateral position after exercise A moderately loud diastolic murmur may persist when the clinical and physiologic results are excellent Comparison of the murmurs before and after commissurotomy in a group of 50 patients showed that the intensity of the murmur decreased by a little over one grade that is if the murmur was of grade 3/6 intensity it became somewhat less than a grade 2/6 The better results were usually associated with a greater decrease in intensity Many of the patients were examined at a time when the valve was not opened as widely as possible and perhaps the decrease in intensity now obtained would be somewhat greater The fact remains however that a significant diastolic murmur usually persists This is possibly due to some residual stenosis and to a loss of flexibility of the valve An increased flow through the opened valve may tend to produce more murmur If the valve becomes stenosed again the murmur increases in loudness Since a residual murmur is usually present before stenosis recurs it is difficult to assess the degree of stenosis on the basis of the murmur alone

A tight mitral valve without mitral insufficiency may be operated upon without the production of any mitral insufficiency Not infrequently however a faint or moderately loud murmur of mitral insufficiency will be heard after commissurotomy when none was present before The murmur usually persists but may become less intense over a period of time A murmur of mitral insufficiency that is present before operation usually is present after operation and may be of the same or of increased intensity

TRICUSPID COMMISSUROTOMY —The common error is overestimation of the degree of tricuspid stenosis Even when the signs of tricuspid stenosis are marked the valve is rarely less than 2 sq cm in area If a loud systolic murmur of tricuspid insufficiency is pres

ent the diagnosis of tricuspid stenosis should be made with caution The middiastolic murmur should be loud and the systolic murmur of tricuspid insufficiency should not be too loud

AORTIC STENOSIS —Aortic stenosis resulting from rheumatic fever is associated almost always with some mitral valve involvement and usually with some degree of aortic insufficiency The problem arises whether in a patient who is being operated upon for mitral stenosis the degree of aortic stenosis is such as would make the operation on the aortic valve worth while In making the decision one finds the loudness of the murmur of aortic stenosis and the comparative loudness of the stenotic and insufficiency murmurs to be of limited help and they must be considered together with evidence obtained by other methods of examination

AORTIC INSUFFICIENCY —The murmur can of course make the diagnosis but peripheral signs of aortic insufficiency and cardiac size are more valuable when one is making the decision regarding the severity of the condition and the need for operation If the peripheral signs of aortic insufficiency are marked the importance of a loud systolic murmur should be minimized

CHAPTER 13

Auscultatory Phenomena in Arteriosclerosis, Hypertension, and Syphilis

ARTERIOSCLEROSIS AND CORONARY HEART DISEASE

A SYSTOLIC MURMUR in the aortic region is a common finding and should be distinguished from the other basal systolic murmurs (p 81)

Apical systolic murmurs are not as frequently heard but often present a diagnostic problem

1 Many are transmitted from the aortic region If the quality of the murmur is similar in the two regions the murmur should be considered as coming from the aortic region This may be true even if the murmur is loudest at the apex because the sounds in the aortic region in older people are diminished by an increase in the diameter of the chest or by emphysema

2 Many apical systolic murmurs are of rheumatic fever origin The patient may admit upon questioning that he has known about the murmur for many years I have been impressed by the number of times a systolic murmur previously unknown and unheard in an older person has on autopsy turned out to have been due to mild rheumatic mitral involvement

3 The murmur should be carefully checked to see if it is actually of maximum intensity at the apex Sometimes the murmur will be found to be of maximum intensity midway between the

apex and the sternal border Such a murmur may perhaps be produced like the innocent murmurs by some change in flow rather than by any valvular involvement

4 Arteriosclerotic involvement of the mitral valve or mitral annulus may sometimes be a cause of the murmur

5 If the heart involvement is sufficient to produce increase in cardiac size with left ventricular dilation the murmur may be due to a relative mitral insufficiency This murmur and that described under (2) above ought to have a higher pitch than the other murmurs

ACUTE MYOCARDIAL INFARCTION

When the extent of infarction is sufficient to affect the functional capacity of the heart the heart sounds are often diminished with the first heart sound more affected than the second heart sound A pericardial friction rub occurs in many cases Auscultation may reveal various arrhythmias An apical systolic murmur may develop during the first few weeks after an infarction since it is unlikely that this murmur is produced by valvular involvement the following possibilities are suggested

1 A relative mitral insufficiency from increase in size of the left ventricle

2 A weakening by infarction of one of the papillary muscles permitting the cusp to balloon into the left ventricle

3 Rupture of some of the chordae tendineae resulting in a mitral insufficiency

4 A weakening of the muscle around the mitral valve with an inability of the valve ring to contract properly

The following causes of a suddenly occurring murmur in acute myocardial infarction can often be recognized

1 Rupture of a papillary muscle This occurs most commonly with a posterior myocardial infarction and the posterior papillary muscle is usually involved The murmur is maximum at the apex and tends to be loud and harsh There is usually no thrill Severe pulmonary congestion sets in quickly

2 Rupture of the interventricular septum The murmur is at the lower end of the sternum and may develop suddenly or gradually The intensity of the murmur as well as the clinical course of the patient will vary with the size of the lesion The lesion occurs usually with anteroseptal infarctions

CARDIAC DECOMPENSATION

1 The murmur of mitral insufficiency may develop as a consequence of dilation of the left ventricle This is a relative mitral insufficiency

2 The pulmonic second sound increases in intensity and becomes louder than the aortic second sound (p 43)

3 Very occasionally a faint early diastolic murmur of pulmonary insufficiency may develop (p 102)

4 The systolic murmur of tricuspid insufficiency can occur as a result of right ventricular dilation (p 74)

5 The first sound may be faint and prolonged and is not usually accentuated

6 A gallop rhythm may occur (p 57)

7 In some patients with cardiac decompensation and a large left ventricle an apical middiastolic murmur will be heard (p 102)

HYPERTENSION AND HYPERTENSIVE HEART DISEASE

1 The aortic second sound is accentuated (p 43)

2 A systolic murmur may occur in the aortic region (p 80)

3 In a few patients the early diastolic murmur of aortic insufficiency may be heard (p 93)

4 An apical systolic murmur may result from left ventricular enlargement and relative mitral insufficiency (p 72)

CARDIOVASCULAR SYPHILIS

1 A systolic murmur may result from dilation of the aorta (p 80)

2 A tympanitic and accentuated aortic second heart sound is heard with aortic dilation

3 The diastolic murmur of aortic insufficiency (p 93) results from dilation of the aortic ring and widening of the commissures This murmur may become very loud

4 With marked aortic insufficiency a systolic murmur is present in the aortic region This murmur is produced by the dilation of the aorta and the increased velocity of blood flow through the aortic valve The murmur may be loud and suggest the murmur of aortic stenosis The aortic second sound may be masked and difficult to recognize especially at the base

5 A middiastolic and presystolic murmur may be heard as the left ventricle dilates (p 98)

6 An apical systolic murmur is usually present and may be transmitted from the aortic region or be the result of a relative mitral insufficiency

7 A loud, very musical diastolic murmur occurs in some patients in whom a cusp becomes retroverted (p 96)

The auscultatory findings in some patients with cardiovascular syphilis may be indistinguishable from those heard in patients with rheumatic heart disease with combined mitral and aortic involvement

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